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Emotion Regulation, Executive Functioning and Quality of life following stroke

A research portfolio

Mhairi Yule

Submitted in part fulfilment of the degree of Doctorate in Clinical Psychology

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Abstract

Systematic review

Executive dysfunction is commonly reported following stroke with most research in this area focused on frontal lobe lesions. A systematic review was carried out to evaluate the evidence of executive dysfunction following stroke as compared to control groups. It was found that executive functions are consistently impaired following stroke and is not limited to frontal lobe lesions. Processing speed, mental flexibility, attention and working memory impairments were found to be the most common executive functioning impairments following stroke. Given the impact executive dysfunction may have on successful rehabilitation, relationships, return to work and quality of life, a comprehensive assessment of such difficulties is important following stroke to aid in the development of appropriate and effective rehabilitation strategies. Future research should use larger samples and a wide range of measures to assess different aspects of executive functioning.

Introduction

Mood disorders and psychological distress are common following stroke, and depression and emotional lability have been studied extensively. There has, however, been little research into difficulties in emotion regulation following stroke and whether this is associated with emotional or cognitive difficulties. The current study investigated emotion regulation difficulties following stroke and their relationship with quality of life, executive functions, anxiety and depression.

Method

Fifty participants who had suffered a stroke and forty five age matched controls completed the Difficulties in Emotion Regulation Scale, the Hospital Anxiety and Depression Scale, the World Health Organization Quality of Life assessment – Brief version and the National Adult Reading Test. In addition individuals with stroke completed four measures of executive functioning – Color Trails Test, Verbal Fluency, Brixton Spatial Anticipation Test and the Hayling Sentence Completion Test.

Results

Individuals with stroke had significantly greater difficulties in emotion regulation compared to age matched controls and this was significantly associated with lower self reported quality of life and increased levels of both anxiety and depression. No significant associations were found between emotion regulation and executive functions or between executive dysfunction and lower self reported quality of life.

Discussion

The current study found evidence that individuals who have suffered a stroke have more difficulties in emotion regulation than an age matched control group. This is clinically important as emotion regulation difficulties are found in mood disorders and it may be that such difficulties precipitate and/or maintain depression following stroke. The absence of an association between difficulties in emotion regulation and executive functioning suggests that other factors may influence such difficulties, such as the trauma of having a stroke. Future research should explore emotion regulation difficulties further following stroke, particularly investigating whether the course of these difficulties changes over time and if they are linked to type of stroke or lesion location.

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Part I: Systematic Review

Chapter one: Systematic Review

Executive dysfunction following stroke: A systematic review

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This review has been written in accordance with ‘The Clinical Neuropsychologist’

Guidelines (Appendix 1)

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Abstract

Background:

Executive dysfunction is commonly reported following stroke with most studies focused upon participants with frontal lobe lesions. A growing body of evidence indicates that executive dysfunction may occur following stroke in the absence of frontal lobe lesions and thus may affect successful rehabilitation following lesions that affect a wider variety of brain locations.

Objectives:

A systematic review was undertaken to examine the evidence for executive dysfunction in stroke patients compared to control groups to find out whether particular aspects of executive functioning are impaired following stroke.

Results:

Impairment in executive functioning following stroke is not limited to frontal lobe lesions and has been found in individuals who have stroke lesions affecting the basal ganglia and cerebellum. Processing speed, mental flexibility, attention and working memory are the most common executive functioning impairments found following stroke.

Conclusion:

Impairment in aspects of executive functioning is common following stroke and found in those without substantial frontal pathology. Rehabilitation techniques for executive impairments following frontal stroke may therefore have utility for a wider range of individuals who have had a stroke, including those with non frontal lesions. A comprehensive assessment of executive functioning is important following all types of stroke given the impact executive dysfunction has on return to work, social relationships, quality of life and other aspects of cognitive functioning.

Introduction

Neuropsychological deficits are common following a stroke (Barker-Collo, Feigin, Parag, Lawes & Senior, 2010). Haring (2002) reports that there is no consistent cognitive profile post stroke, with the most common impairments in memory, visuo-perceptual abilities, apraxia, executive functions and attention (Lincoln, Kneebone, Macniven & Morris, 2012). The current review focuses on executive dysfunction following stroke.

Barker-Collo et al. (2010) found that, five years post stroke, aspects of executive functions and processing speed were the greatest cognitive impairments, however, there is conflicting evidence for the role of different types of stroke and lesion location in executive dysfunction. Vataja, Phojasvaara, Mantyla, Ylikoski, Leppavouri et al. (2003) found that lesions affecting frontal-subcortical connections resulted in executive dysfunction, however, Park, Yoo and Rhee (2011) found executive dysfunction following posterior cerebral artery stroke. Left hemisphere stroke has also been found to lead to greater impairments in executive functions than right hemisphere stroke (e.g. Vataja et al. 2003, Hermann, Siccoli, Brugger, Wachter, Mathis et al. 2007). Tang, Chan, Lam, Mok, Wong et al. (2009) did not find a correlation between executive dysfunction and either frontal or basal ganglia lesions which is in contrast to other research, however, this provides support for the theory that areas other than the frontal lobes are involved in executive functions.

Definitions of Executive Functions

Executive Functioning is an umbrella term used to refer to a number of different cognitive abilities and behaviours. It is the ability to engage in “independent, purposive, self serving behaviour” (Lezak, Howieson & Loring, 2004; p35) and involves a number of cognitive domains including planning, problem solving, set shifting, initiation, concept formation, reasoning and mental flexibility (Hodges, 1994). The literature on executive functions uses a number of different terms, however, they are generally understood as higher level cognitive functions exerting control over lower cognitive functions (Stuss & Levine, 2002) rather than as a single, discrete cognitive function (Kudlicka, Clare & Hindle, 2011). The two predominant theories of executive functions are the Working Memory Model (Baddeley, 2000) and The Supervisory Attentional System (Shallice, Burgess & Robertson, 1996). The Working Memory Model consists of the visuospatial sketchpad for processing visual information, a phonological loop for processing language, an episodic buffer and a central executive that allows information to be manipulated (Baddeley, 2000). Goldman-Rakic

(1998) argues that the central executive of the Working Memory Model may be multiple sub processes rather than one processor and is thought to be related to the Supervisory Attentional System (Evans, 2007). The Supervisory Attentional System uses incoming perceptual information to trigger a response to routine or novel situations based on existing schemas (Shallice, Burgess & Robertson, 1996). This system is required for planning, error correction, responses to novel or dangerous situations and situations requiring overcoming a habitual or impulsive response (Evans, 2007). Traditionally, these systems were hypothesised to be linked to frontal lobe function (Hodges, 1994) due to case studies of frontal lobe damage resulting in difficulties with executive functions (e.g. Grafman, Sirigi, Spector & Hendler, 1993; Damasio, Grabowski, Frank, Galaburda & Damasio, 1994). These difficulties can however, occur following damage to other areas of the brain (Lincoln et al. 2012).

Dysexecutive syndrome was suggested as a functional description of cognitive difficulties involving goal directed behaviour, with the use of this term aimed at reducing the focus on localisation inherent in terms that refer to the frontal lobes. The clinical features of dysexecutive syndrome are the inability to initiate, stop and modify behaviour, difficulties organising, planning and problem solving, difficulty inhibiting responses, perseveration and impaired working memory (D'Esposito & Gazzaley, 2006). A breakdown at any point in the Supervisory Attentional System can result in the same observable difficulty or consequence for different reasons (Evans, 2007). There may be difficulties with some sub processes whilst others remain intact. This can lead to impairments in attention and, in turn; distractibility resulting in goal neglect. Difficulties sequencing a task may also lead to goal neglect for different reasons (Evans, 2007). This model is a functional rather than anatomical model of executive functioning which may provide further support that executive functions are not exclusively related to frontal lobe damage.

The basal ganglia are made up of subcomponents including the caudate nucleus, lentiform nucleus, subthalamic nucleus and the substantia nigra (Herrero, Barcia & Navarro, 2002). The striatum receives input from the cortex and is connected to the frontal lobes through the thalamus (Elliot, 2003). These areas are important for the motor system (Herrero *et al.* 2002) however, single case studies following subcortical stroke (Rainville, Firoire, Periot, Cuny & Mazaux, 2003) and basal ganglia stroke (Swainson & Robbins, 2001) have found evidence of executive dysfunction. Subcortical conditions such as Parkinson's Disease which are predominantly affected by motor impairment (Elliot, 2003) have been shown to

present with impairments in executive functions (Kudlicka, Clare & Hindle, 2011). Cerebellar stroke (Paulus, Magnano, Conti, Galistu, D'Onofrio et al. 2004) has also been found to result in executive dysfunction. This provides further evidence that damage to the frontal lobes is not the only mechanism leading to executive dysfunction and that other areas or systems are implicated. In individuals with lesions primarily affecting areas of the brain other than the frontal lobes, it is likely that blood supply to other brain areas will also be affected (Lincoln et al. 2012). For example, occlusion of the middle cerebral artery may affect blood flow to the frontal lobes as well as other areas of the brain which may go some way toward explaining the wide range of cognitive impairments that may be experienced post stroke.

Measurement issues in executive functions

As the definition of executive functioning varies across studies, it is often clear that different studies are referring to different constructs and may be measuring single constructs that make up executive functions rather than multiple components. The term 'frontal functioning' has often been used interchangeably with executive functioning which may foster the belief that only damage to the frontal cortex leads to these impairments. Stuss and Levine (2002) describe executive dysfunction as the most common presentation in neuropsychological practice and therefore it is important that neuropsychological assessments are able to detect these deficits. However, patients with 'real world' executive functioning difficulties may perform well on many neuropsychological assessment tasks due, in part, to the structured environment in which such assessments are usually carried out (Lezak et al. 2004) both in clinical and research settings. Traditional neuropsychological assessments also may not measure certain aspects of executive functions (Lezak et al. 2004).

There are a number of neuropsychological assessments hypothesised to assess different areas of executive functioning and, latterly, assessment batteries such as the Behavioural Assessment of the Dysexecutive Syndrome (BADS; Wilson, Alderman, Burgess, Emslie & Evans, 1996) and the Multiple Errands Test (Knight, Alderman & Burgess, 2002) have been developed as more ecologically valid measures of executive functions. However, test selection is a matter of ongoing controversy and there is no agreed upon battery for assessment of executive functions (Kudlicka et al. 2011). Many neuropsychological assessments measure reasoning and problem solving abilities rather than social and emotional skills which may reflect why patients with dysexecutive syndrome may perform well with traditional assessments yet struggle with real life tasks. It is hypothesised that the

dorsolateral prefrontal cortex is involved in reasoning and problem solving abilities and the orbitofrontal cortex is involved in social and emotional skills (Fuster, 2001) and damage to these areas results in the dysexecutive syndrome.

Rationale for systematic review

There is evidence that executive dysfunction following stroke can affect physical rehabilitation and functional outcomes (Zinn, Bosworth, Hoenig & Swartwelder 2007). Executive dysfunction following stroke may also play a role in recovery and have an effect on level of dependence and return to work (Poulin, Korner-Bitensky, Dawson & Bherer, 2012). It is important that executive functioning impairments are detected following stroke in order to develop appropriate goals for rehabilitation and to assess the impact of these deficits on other cognitive functions. The nature and extent of specific deficits will lead to different intervention plans and goals. Research to date has found conflicting evidence regarding the evidence for executive dysfunction post stroke and whether it is associated with particular types of stroke. The purpose of this review is to explore further the evidence for executive dysfunction following stroke.

Method

Review objective

The objective of the review is to evaluate the evidence for executive dysfunction in individuals with stroke.

Search strategy

The following search terms were used in this study: “stroke”, “cerebrovascular”, “executive”, “dysexecutive” and “frontal lobe”. The following electronic databases were searched until 29th May 2012: MEDLINE (from 1946), PsychINFO (from 1806) and EMBASE (from 1947).

Inclusion/exclusion criteria

Criteria for the review were developed using the PCOS (Population, Comparators, Outcomes, Study Design) format described by the Centre for Reviews and Dissemination (Centre for Reviews and Dissemination, 2008) and the Scottish Intercollegiate Guidelines Network (SIGN 50, 2011).

Population

Studies were included where at least one subcomponent of executive functioning was measured as part of a study investigating cognitive impairment in human adults post stroke. Studies were excluded if they included patients with other brain injuries or degenerative conditions unless stroke participant data were presented separately. Studies investigating subarachnoid haemorrhage were excluded as this may show a different pattern of impairment due its nature and different underlying pathology. Any studies investigating cerebrovascular disease, small vessel disease or dementia were excluded as the purpose was to investigate executive functions following stroke and these conditions are considered progressive and have different aetiologies.

Comparators

Studies that did not compare individuals with stroke to a control group with out stroke or condtions known to impact on neuropsychological functioning were excluded.

Outcomes

Executive functioning was the outcome of interest; therefore, only studies that included at least one measure of executive functioning were included.

Study Design

Included studies were in English and presented original data. Conference abstracts and book titles were excluded. Studies were also excluded if executive functioning results were not included separately.

Quality Criteria

Quality criteria were developed in order to determine the robustness of each study in relation to the review question. These were developed using the Scottish Intercollegiate Guidelines Network methodology checklist three: cohort studies (SIGN, 2011). These guidelines cover four areas: selection of participants, assessment, confounding variables and statistical analyses. These guidelines were adapted to the needs of the current review and are outlined in Table 1.1. The original criteria are contained in appendix two. Sherer, Novak, Sander, Struchen, Alderson et al. (2002) developed guidelines for assessing the methodological quality of neuropsychological research and propose that a good study should take confounding factors into account, use norms, use outcome measures related to outcome of interest, describe recruitment and eligibility criteria, use an adequate sample size and appropriate statistical analyses. When the guidelines for the current review were developed this guidance was used to assist in tailoring the SIGN guidelines as appropriate for the aims of the current review.

In the current review, with regard to participants, the quality criteria used were matched control groups to minimise confounding variables, specification of eligibility criteria and sample size. These were relevant to answering the review question and are recommended by Sherer et al. (2002). With regard to criteria relating to assessment, blinding was used as a quality criteria, as it may be that examiner knowledge of a participant's group membership (stroke or control) would affect expectations and assessment scoring. Validity and reliability of the executive functioning measures was also used as if conclusions are to be drawn regarding performance on these measures there must be sufficient evidence of their quality. With regard to confounding variables, the criteria relating to matching the control group covers this criterion. To address statistical analyses, criteria were used in relation to use of appropriate statistical analyses as recommended by Sherer et al. (2002). A criterion was also used for reporting of effect sizes as a significant result may have a small effect size and therefore limited clinical utility, therefore effect sizes were thought to be appropriate for addressing the review question.

Table 1.1: Quality criteria for evaluating methodological quality of included studies

1. Study addresses appropriate, clearly defined question	Research questions are clearly defined and appropriate	Well covered (3)
	Research questions adequately defined and appropriate	Adequately addressed (2)
	Research questions insufficiently defined	Poorly addressed (1)
	Questions are not specified	Not addressed/not reported (0)
2. Validity and reliability of executive functioning measures used	Standardised measures of executive function used. Measures well validated and reliable and cover more than one area of executive function. All measures have evidence of good validity and reliability.	Well covered (3)
	Standardised measures of executive function used. Measures well validated and reliable and cover one area of executive function. At least 50% of these measures have evidence of good validity and reliability.	Adequately addressed (2)
	Non standardised measures of executive function used or measures of executive function that are not well validated and reliable were used. Less than 50% of the measures have evidence of good validity or reliability.	Poorly addressed (1)
	No valid or reliable measures used	Not addressed/not reported (0)

3. Matched control group to minimise confounding variables	Groups matched in terms of age and education or differences are controlled for in analysis. Two groups comparable on important variables.	Well covered (3)
	Only well matched or suitably controlled in terms of either age or education e.g. only matched for age	Adequately addressed (2)
	The control group is not well matched to the stroke group in terms of age and education and these differences are not controlled for in analyses	Poorly addressed (1)
	Groups not matched on key demographic variables or there is no data reported on whether the control group is matched to the stroke group	Not addressed (0) Not reported (0)
4. Sample size	Number of participants sufficient to enable power of 0.8 for medium effect size and alpha 0.5 for comparisons between stroke and control participants	Well covered (3)
	Number of participants sufficient to enable power of 0.7 for medium effect size and alpha 0.5 for comparisons between stroke and control participants	Adequately addressed (2)
	Number of participants was only sufficient to enable power of less than 0.7 for medium effect size and alpha 0.5 for comparisons between stroke and control participants	Poorly addressed (1)
	Sample size not reported	Not reported (0)

5. Eligibility criteria specified	Clearly defined inclusion criteria, including all of the following: type and severity of stroke, time since stroke, whether participants have any other difficulties (e.g. aphasia), whether all participants had only one stroke or whether the study included those who had more than one stroke and control group information clearly defined.	Well covered (3)
	Meets all but one of the following: type and severity of stroke, time since stroke, whether participants have any other difficulties e.g. aphasia, number of strokes of participants detailed e.g. whether all participants had only one stroke or whether included those who had more than one stroke and control group criteria clearly defined.	Adequately addressed (2)
	Some information is provided on inclusion and exclusion criteria but it is not clear, no consideration given to type/severity of stroke and time since stroke. Would be difficult to replicate study on basis of details given. No clear criteria for control group.	Poorly addressed (1)
	No information provided on any of the criteria	Not addressed (0)

6. Examiners blind to whether participant is from stroke or control group	Examiners were unaware whether participants were from the stroke group or the control group when assessing and scoring executive functioning measures	Well covered (3)
	Examiners were unaware whether participants were from the stroke group or the control group when assessing or scoring executive functioning measures	Adequately addressed (2)
	Some efforts were made to ensure examiners were unaware of which group participants belonged to, however, this was not consistent throughout the study.	Poorly addressed (1)
	Examiner awareness of participant group not reported or not described clearly enough to work out	Not addressed (0) Not reported (0)
7. Appropriate analysis used for measures and study design	Analysis used is clearly appropriate to address the research question including consideration of statistical methods used to deal with missing data.	Well covered (3)
	Analysis used seems appropriate, including consideration of statistical methods used to deal with missing data, but is less well described	Adequately addressed (2)
	Analysis used seems inappropriate to address the research question and / or there are inadequate methods for dealing with missing data.	Poorly addressed (1)
	Analysis is inappropriate or not reported	Not addressed (0) Not reported (0)

8. Effect sizes reported for executive function measures	Effect sizes reported for all tests or could be calculated from data presented.	Well covered (3)
	Effect sizes reported for at least 50% of the tests or could be calculated from data presented.	Adequately addressed (2)
	Effect sizes reported for less than 50% of the tests	Poorly addressed (1)
	Effect sizes not reported and data not sufficient to be calculated.	Not addressed (0)

Results

Study inclusion

The search terms yielded 1619 results (excluding duplicates). Titles and abstracts were screened and those which were clearly irrelevant were excluded. This resulted in 1431 papers being excluded. Abstracts of remaining studies were screened and those not meeting the inclusion and exclusion criteria were also excluded as illustrated in Figure 1.1.

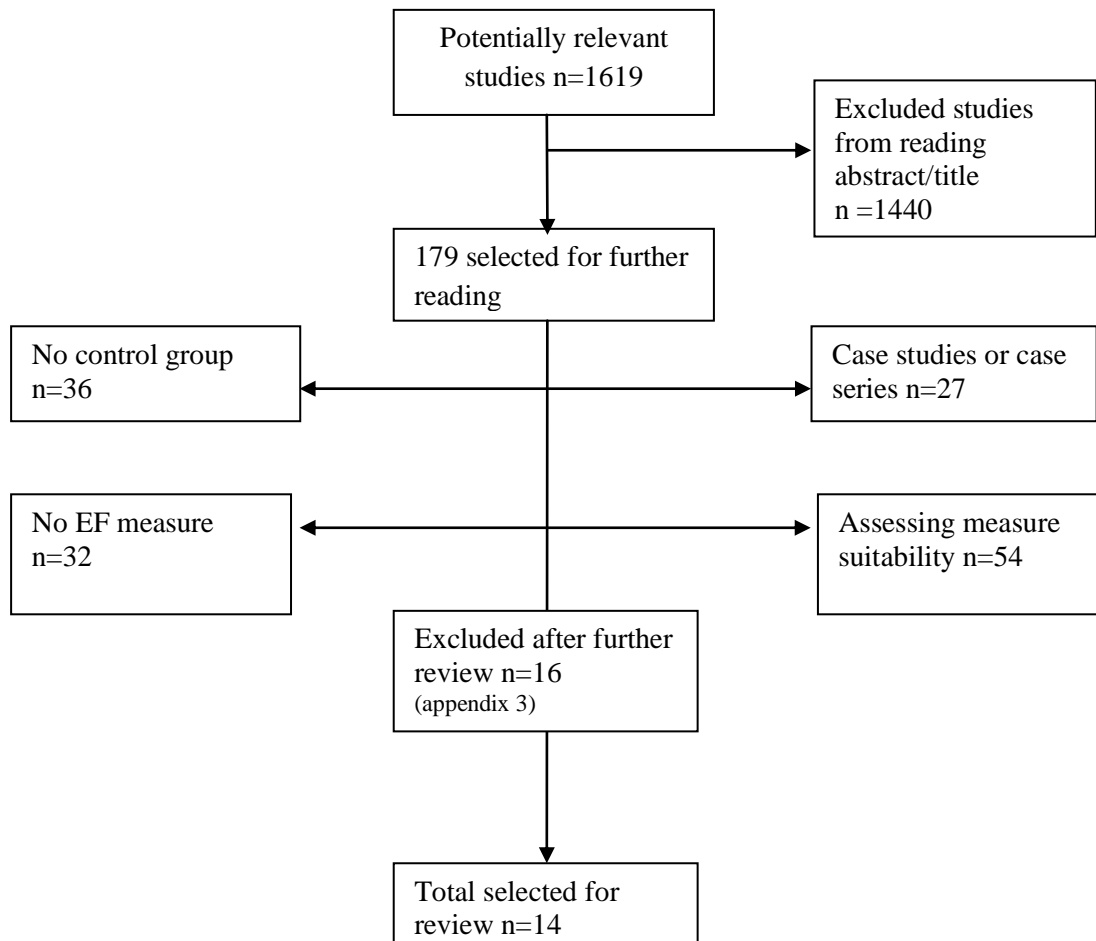


Fig 1.1: Identified literature and reasons for exclusion

General characteristics of included studies

A summary of the 14 included articles is provided in Table 1.2. All of the included studies were cross sectional studies. Seven of the included papers evaluated general neuropsychological functioning following stroke (Engstad et al. 2003; Planton et al. 2012; Su et al. 2007; Stephens et al. 2004; Rao et al. 1999; Knopman et al. 2004, Sachdev et al. 2003) and seven were particularly focused on executive dysfunction following stroke (Cools et al. 2006; Manes et al. 2006; Nys et al. 2006; Eslinger & Grattan 1993; Zinn et al. 2007; Leskela et al. 1999; Godefroy et al. 2010). Two studies compared frontal versus striatal lesions, (Cools et al. 2006; Eslinger & Grattan, 2003) one investigated cerebellar lesions (Manes et al. 2009), one investigated basal ganglia haemorrhage (Su et al. 2007) and ten did not focus on particular types of stroke. The number of participants with a stroke in the studies varied from 10 (Zinn et al. 2007) to 381 (Stephens et al. 2004). One study used a computer based switching task (Cools, Ivry & D'Esposito, 2006) and all others used published and standardised tests of executive function. Two studies used only one test of executive functioning (Su et al. 2007; Cools, Ivry & D'Esposito, 2006).

Quality assessment

In order to evaluate the methodological strength of the included studies, the quality criteria outlined in Table 1.1 were applied to each of the studies, with quality ratings presented in Table 1.3.

Table 1.2: Studies included in systematic review

Study	Aims	Participants	Executive Function Measures	Key results	Study conclusions
Engstad, Almkvist, Viitanen, & Arnesen (2003)	To compare cognitive function in long term stroke survivors to a control group. To find out which neuropsychological tests best identify those with and without stroke.	Stroke group: n=198 57.6% male Mean age 68.1 Tested on average 8.9 years following stroke. Control group: n=242 55.8% males Mean age 65.1	Verbal fluency TMT B Digit symbol test	Individuals in the stroke group had significantly lower scores than those in the control group on all executive function measures ($p < 0.01$).	Verbal fluency significantly differentiated individuals with stroke from control participants. Test scores declined with increasing age.
Cools, Ivry & D'Esposito (2006)	To compare individuals with frontal and striatal ischaemic lesions to neurologically healthy individuals on a cognitive switching task.	Frontal stroke group: n=6 4 males Mean age 53.0 Tested from 1 to 20 years post stroke. Striatal stroke group: n=6 4 males Mean age 61.5 Control group: n=6 2 males Mean age 61.0	Computer task involving cognitive switching	Striatal lesion group made disproportionately more errors on stimulus switch than rule switch trials. Frontal lesion group and control group made more errors on rule switch than stimulus switch. Deficit in switching in individuals with focal striatal lesions but not in individuals with frontal lesions.	The striatum is necessary for cognitive flexibility. The prefrontal cortex is involved in but not necessary for the type of cognitive switching used in this study.
Manes, Villamil, Ameriso, Roca & Torralva (2009)	To investigate executive functions in patients with cerebellar stroke and to assess real life executive deficits.	Cerebellum stroke group: n=11 9 males Mean age 50.6 Tested from 6 months post stroke. Control group: n=6 No information but states participants matched on age ($p > .05$)	Verbal fluency Backward digit span TMT A&B WCST Test of Attentional Performance MET	Individuals with stroke were significantly poorer on semantic fluency, WCST, flexibility subscale of Test of Attentional Performance. On MET, those with stroke significantly poorer on four of five subscales (total failures, aim interpretation, fulfilment of tasks and inefficiencies).	Focal cerebellar lesions can impair real life problem solving and executive function assessments.

Planton, Peiffer, Albucher, Barbeau, Tardy et al (2012)	<p>To assess neuropsychological outcome following first ischaemic stroke.</p> <p>To identify profile of impairment and relations between lesion location and outcome.</p>	<p>Stroke group: n=60 65% male Mean age 59.7 Average 109 days post stroke.</p> <p>Control group: n=40 35% male Mean age 57.2</p>	<p>TMT A&B, Stroop Digit symbol TEA digit span spatial span verbal fluency MCST</p>	<p>Individuals with stroke significantly poorer than control group on all measures.</p> <p>Large effect size for speed of processing, initiation, working memory and continuous attention.</p> <p>No significant differences between cortical, subcortical and intratentorial subgroups or between left and right infarcts.</p>	<p>There was evidence for impairments in executive functions following stroke but further study needed to better characterise this.</p> <p>Impairments were not specific to type of stroke or hemisphere of stroke.</p>
Su, Chen, Kwan, Lin & Guo (2007)	<p>To determine the severity and pattern of cognitive dysfunction in basal ganglia haemorrhage.</p>	<p>Basal ganglia group: n=30 83.3% male Mean age 53.8</p> <p>Control group: n=37 43.2% male Mean age 56.9</p>	<p>WCST</p>	<p>Significant between group effect for executive function.</p> <p>Those with right side lesions performed significantly worse than those with left side lesions.</p>	<p>The scores of individuals with Basal ganglia haemorrhages were significantly lower than the control group for executive functions, particularly for right hemisphere haemorrhages.</p>
Stephens, Kenny, Rowan, Allan, Kalaria et al (2004)	<p>To compare individuals with stroke and no cognitive impairment (NoCIND), stroke and vascular cognitive impairment (VCIND) and stroke with vascular dementia (VaD) to a control group to find predictors of further decline.</p>	<p>No cog impairment: n=259 55.6% males Mean age 80.31</p> <p>Vascular cog impairment: n=92 42.4% males mean age 81.61</p> <p>Vascular dementia: n=33 55.6% males mean age 79.57</p> <p>Control group: n=66 58.8% male Mean age 80.47</p> <p>Assessed 3 months post stroke.</p>	<p>CAMCOG-R CDR computerised battery (simple reaction time, choice reaction time, number vigilance task)</p>	<p>No CIND group were more impaired than control group on all but one cognitive measure, most notably impaired on executive functions, processing speed & perception.</p> <p>VCIND group were more impaired on all measures compared to NoCIND, particularly memory, executive functions and language.</p> <p>VCIND group were comparable to VaD particularly on executive functions & abstract thinking.</p>	<p>Attention and executive functioning deficits found in NoCIND group. These deficits were worsened in the VCIND group.</p> <p>Attention and executive functioning impairments are characteristic of individuals who have had a stroke.</p>

Rao, Jackson & Howard (1999)	To assess neuropsychological impairment associated with cerebrovascular disease and peripheral vascular disease (PVD).	Anterior circulation stroke group: n=25 9males Mean age 74.9 Assessed 6 months to 1 year post stroke Carotid stenosis & TIA group: n=25 15 males Mean age 75.7 PVD group: n=25 20 males Mean age 72.9 Control group: n=25 9 males Mean age 74.9	CAMCOG TMT BDS COWAT	Individuals with stroke were significantly lower than the control and TIA groups in abstract thinking, attention and verbal fluency. Individuals with stroke and TIA's were significantly poorer than the control group on the BDS. Individuals with stroke were more impaired than those with TIA's on TMT A and were more impaired than all groups on TMT B.	Those with anterior circulation stroke were poorer on initiation, mental flexibility & set shifting than those with TIA's, PVD and the control group.
Nys, Van Zandvoort, van der Worp, Kapelle & de Haan (2006)	Study 1: To examine prevalence and neuropsychological correlates of perseverative response in individuals with stroke Study 2: To determine neuroanatomical correlates of perseverative responses following acute stroke Participants with stroke separated into four groups according to level of difficulty with neglect and compared to a control group.	No neglect group: n=140 52.9% male Mean age 60.9 General inattention group: n=31 29% Male Mean age 65.8 Left neglect group: n=23 60.9% Males Mean age 66.4 Right neglect: n=12 46.2% Males Mean age 68.1 Control group: n=63 33.3% Males Mean age 61.5	Visual elevator (from TEA) Verbal fluency	All patient groups performed significantly worse than healthy controls on tests of executive function ($p < 0.001$). There was an association between perseveration and executive dysfunction in stroke patients without inattention. Disturbed working memory and executive dysfunction were related to inattention but not to perseverative responses.	Basal ganglia stroke is associated with perseveration. Hemisphere of lesion is not associated with perseveration.

Eslinger & Grattan (1993)	To explore whether frontal and striatal areas mediate distinctive or overlapping aspects of cognitive flexibility.	Frontal stroke group: n=10 8 males Mean age 65.2 Basal ganglia stroke group: n=10 4 males Mean age 64.1 Posterior stroke group: n=10 5 males Mean age 53.1 Control group: n=10 Mean age 61.6	WCST Alternate Uses Test	WCST: Frontal and basal ganglia stroke groups made significantly more errors than the posterior stroke group and control group. AUT: Frontal stroke group significantly poorer than all other groups. Basal ganglia group not significantly different from posterior group and posterior stroke group not significantly different from control group.	Frontal and basal ganglia patients did not differ on reactive flexibility part of set shifting. Both the frontal and basal ganglia group made significantly more perseverative errors than the posterior group and controls. Spontaneous flexibility more impaired in frontal lesions than basal ganglia or posterior lesions.
Zinn, Bosworth, Hoenig & Swartzwelder (2007)	To investigate the frequency of executive dysfunction in acute stroke and to explore the relationship between this and stroke severity.	Stroke group: n=47 Mean age 65.8 Average 4.6 days post stroke TIA group: n=9 Mean age 64.1 At risk for stroke: (control) n=10 95% male Mean age 58.5	Digit span & picture arrangement (WAIS – III) Symbol digit modalities Test, design fluency & trail making (D-KEFS)	Individuals with stroke were impaired on 60% of tests. Stroke group were significantly more impaired on symbol digits, trails and design fluency. High rates of executive functioning impairments in non stroke groups. Relationship between EF impairment and premorbid IQ.	Working memory, processing speed and cognitive flexibility most impaired in stroke compared to other groups. Stroke severity not related to EF. EF deficits may exist on a continuum in cerebrovascular disease.
Leskela, Hietanen, Kalska, Ylikoski, Pohjasvaara et al. (1999)	To investigate whether frontal ischaemic stroke leads to executive dysfunction or slowed processing speed.	Frontal stroke group: n=62 32 male Mean age 70.9 Non frontal stroke group: n=188 84 males Mean age 70.3 Control group: n= 39 18 males Mean age 66.5	TMT A and B Stroop WCST Verbal fluency	Frontal stroke group slower on processing speed & EF than control group. Frontal stroke group significantly slower on Stroop and TMT A than non frontal group. Non frontal stroke group performed less well than the control group on processing speed & EF. No significant difference in TMT B & Stroop measuring EF.	Performance is impaired in elderly stroke patients. Those with frontal stroke were slower than those with non frontal stroke in rapid mental processing. Main impairment in frontal stroke was speed of information processing. No specific evidence of dysexecutive syndrome related to frontal lobe lesions.

Godefroy, Azouvi, Robert, Roussel, LeGall & Meulemans (2010)	To investigate the frequency of behavioural and cognitive dysexecutive disorders in different diseases (stroke, TBI, MS, Alzheimers. Parkinsons).	Stroke group: n=152 51% male Mean age 48 Control group: n=461 48% male Mean age 50.5	Behavioural dysexecutive syndrome inventory Cognitive dysexecutive battery	25% of the stroke sample found to have behavioural dysexecutive syndrome. 29% of the stroke sample found to have cognitive dysexecutive syndrome.	Deduction primarily affected in stroke compared to the other medical conditions studied.
Knopman, Roberts, Geda, Boeve, Pankratz et al. (2009)	To explore associations between stroke history, APOE genotype and subtypes of mild cognitive impairment (MCI).	Stroke group: n=183 51% male MCI group: n= 273 Control group: n= 1513 (no stroke or MCI) All participants aged 70 to 89	TMT B Digit symbol substitution (WAIS –R)	History of stroke was associated with MCI. History of stroke associated with impairment in all cognitive domains except memory – magnitude strongest for executive function. In ‘cognitively normal’ participants, stroke was associated with lower performance in EF.	Executive dysfunction found in those with stroke with no prior evidence of cognitive impairment.
Sachdev, Valenzuela, Brodaty, Wang, Looi et al. (2003)	To investigate whether homocysteine levels are a risk factor for cognitive impairment after a stroke – language, memory, attention and executive functioning.	Stroke group: n=95 ischemic stroke Mean age 72.7 56.6% males 3 months post stroke Control group: n=55 50.6% males Mean age 71.7	TMT A & B Symbol digit WAIS-R similarities WAIS-R picture completion CFS MDRS: Identities and oddities subtest Verbal fluency	Composite z scores computed for each domain. Significant difference in z scores between groups for executive functioning (p<0.001).	Executive functioning impairment is associated with homocysteine levels in stroke.

EF – Executive functions, CVD – Cardiovascular Vascular Disease, TIA – Transient Ischaemic Attack, PVD – Peripheral Vascular Disease, NoCIND – No cognitive impairment, VCIND – Vascular Cognitive Impairment, VaD – Vascular Dementia, WCST – Wisconsin Card Sorting Test, WAIS-R – Wechsler Adult Intelligence Scale Revised, WAIS-III – Wechsler Adult Intelligence Scale, 3rd Edition, MCI – Mild Cognitive Impairment, TBI – Traumatic Brain Injury, MS – Multiple Sclerosis, D-KEFS – Delis-Kaplan Executive Function System, BADS- Behavioural Assessment of the Dysexecutive Syndrome, TEA – Test of Everyday Attention, CAMCOG – Cambridge Cognitive Examination, COWAT – Controlled Oral Word Association Test, CDR – Computerised assessment system, MET – Multiple Errands Test, TMT A– Trail Making Test part A, TMT B – Trail Making Test part B, BDS – Behavioural Dyscontrol Scale, CFS – Colour Form Sort, MDRS-Matthis Dementia Rating Scale.

Table 1.3: Quality assessment criteria

Study	Question	Measure quality	Control group	Sample and power	Eligibility criteria	Blinding	Analysis used	Effect size reported	Quality score
Planton et al (2012)	Well covered	Well covered	Well covered	Well covered	Well covered	Not addressed	Well covered	Well covered	21
Rao, Jackson & Howard (1999)	Well covered	Well covered	Well covered	Adequately addressed	Well covered	Not addressed	Well covered	Not reported	20
Sachdev et al. (2003)	Well covered	Well covered	Adequately addressed	Well covered	Adequately addressed	Not addressed	Well covered	Adequately addressed	18
Su et al. (2007)	Well covered	Adequately addressed	Adequately addressed	Adequately addressed	Well covered	Poorly addressed	Well covered	Not reported	16
Stephens et al (2004)	Well covered	Adequately addressed	Well covered	Well covered	Well covered	Not addressed	Adequately addressed	Not reported	16
Nys et al. (2006)	Well covered	Adequately addressed	Adequately addressed	Well covered	Well covered	Not addressed	Well covered	Not reported	16
Leskela et al (1999)	Adequately addressed	Well covered	Adequately addressed	Adequately addressed	Adequately addressed	Adequately addressed	Well covered	Not reported	16
Engstand et al. (2003)	Well covered	Adequately addressed	Well covered	Well covered	Adequately addressed	Not reported	Adequately addressed	Not reported	15
Godefroy et al (2010)	Adequately addressed	Adequately addressed	Adequately addressed	Well covered	Well covered	Not addressed	Well covered	Not reported	15
Zinn et al. (2007)	Well covered	Well covered	Poorly addressed	Adequately addressed	Adequately addressed	Not addressed	Well covered	Not reported	14
Knopman et al (2009)	Adequately addressed	Adequately addressed	Poorly addressed	Adequately addressed	Well covered	Not addressed	Adequately addressed	Adequately addressed	14
Cools, Ivry & D'Esposito (2006)	Adequately addressed	Poorly addressed	Well covered	Poorly addressed	Well covered	Not reported	Well covered	Not reported	13
Eslinger & Grattan (1993)	Adequately addressed	Adequately addressed	Well covered	Poorly addressed	Adequately addressed	Not addressed	Well covered	Not reported	13
Manes et al. (2009)	Adequately addressed	Well covered	Adequately addressed	Poorly addressed	Adequately addressed	Not addressed	Adequately addressed	Not reported	12

Evidence of executive dysfunction

Of the fourteen studies included in the review, all found evidence of executive dysfunction following stroke as compared to a control group, however, different assessment measures were used across studies and different types of strokes were investigated across studies.

Planton et al. (2012) found that the performance of individuals who had suffered a stroke was significantly poorer than a control group on eight measures of executive function. There was a good sample size with a comparable age range across groups and large effect sizes were reported for processing speed, initiation, working memory and continuous attention. As a number of different tests with good evidence of validity and reliability were used, the conclusions drawn are stronger than studies that used fewer measures that did not cover a range of areas of executive functioning.

Zinn et al. (2007) used five measures of executive functioning and had a large number of participants with a stroke. Working memory, processing speed and cognitive flexibility were found to be significantly impaired as compared to a control group; however, the number of individuals in the control group was less than the number of participants in the stroke group in this study. Sachdev et al. (2003) used six measures of executive function and computed a composite z score for each cognitive domain assessed. Executive functions were found to be impaired in the group of stroke participants as compared to a control group. However, as a composite score was calculated, specific domains of executive functioning were not reported separately.

Rao et al. (1999) and Leskela et al. (1999) each used four executive functioning measures with good evidence of validity and reliability. Leskela et al. (1999) concluded that participants with stroke had impairments in processing speed and executive functions. Examiners in this study were unaware of the neurological or neuroradiological findings when conducting neuropsychological assessments, therefore they could be argued to be less likely to be vulnerable to bias. This is a strength of the study; however, the control group were younger and had a smaller sample size than the stroke group. Executive functions have been found to be poorer with age (e.g. Nguyen et al. 2007) therefore; it could be argued that this was an age rather than a stroke effect.

Rao et al. (1999) compared a group of individuals with stroke to three separate control groups and found that those in the stroke group were poorer on initiation, mental flexibility

and set shifting. As the individuals in the stroke group were compared to age matched individuals with TIA and peripheral vascular disease as well as healthy controls, this is good evidence to support that these impairments are likely to be a result of stroke rather than age related impairments. The three control groups were of a comparable size, however, were small samples.

Stephens et al. (2004) tested participants on the Cambridge Cognitive Examination (CAMCOG) and a computerised battery assessing reaction times and vigilance and concluded that attention and executive functions are impaired following stroke. It could be argued that processing speed is the primary impairment rather than executive functioning per se on the computerised battery. The executive functioning tests on the CAMCOG are ideational fluency and visual reasoning (Leeds, Meara, Woods & Hobson, 2001) therefore this study can only conclude that processing speed, fluency and reasoning are impaired post stroke. The CAMCOG has been found to be vulnerable to the effects of depression (Leeds et al. 2001) and given that post stroke depression is common (Hackett, Yapa, Parag, & Anderson, 2005) it may not be an optimum measure for this population. Importantly, Stephens et al. (2004) found that those with stroke deemed as having no cognitive impairment on clinical diagnosis showed significant impairment in these assessments as compared to a control group.

Nys et al. (2006) compared individuals with stroke with and without neglect and inattention on the visual elevator subtest of the Test of Everyday Attention (TEA) and Verbal Fluency and found all groups performed significantly worse than the control group. However, as these assessments measure attention and verbal fluency, it can not be concluded that all executive functions were impaired as it is not a unitary construct. There were also large discrepancies between the numbers of participants in each group. The individuals in the stroke group with right sided neglect had only 12 participants yet there were 60 controls. This may have led to inability to detect subtle differences between the different groups.

Knopman et al. (2009) used Trail Making part B and Symbol Substitution to assess executive functioning and found impairments on these tasks in stroke participants with no other observable cognitive impairments, however, these tests could be argued to assess processing speed rather than executive functioning per se. The study may have been improved by incorporating a wider range of executive functioning assessments and by using Trail Making part A to control for processing speed. Participants for this study were randomly selected

community residents. History of stroke was obtained from participants themselves and confirmed by medical records resulting in 183 participants with stroke and 1513 participants without stroke; therefore, particular criteria were not specifically applied to the recruitment of stroke participants. Godefroy et al. (2010) recruited participants with stroke as part of a larger study assessing disease and executive functions using a French battery for assessing behavioural and cognitive aspects of executive dysfunction. This study had a mean age of stroke participants as 48; therefore it could be that these results apply predominantly to younger stroke survivors and may not be generalisable.

Su et al. (2007) used the WCST and concluded that stroke participants had significantly poorer executive functioning than the control group however, as only one measure was used, it can only be concluded that aspects of executive functions such as abstraction, concept formation and cognitive flexibility are impaired rather than executive functioning per se. There was also a relatively small sample in this study with a younger age (mean 53.8) than other studies which may affect the generalisability of the results. Different staff carried out assessment for the individuals in each group which may reduce bias; however, blinding may have further reduced this.

In summary, the results of the systematic review show that the most common pattern of executive dysfunction following stroke is in relation to processing speed, working memory, attention and cognitive flexibility. Impairments were also found in initiation, set shifting, fluency and real life problem solving. The studies varied in the measures used and size of samples, however, there appears to be consistent evidence of the above pattern of impairments following stroke as compared with individuals in healthy control samples.

Evidence for executive dysfunction in non frontal versus frontal lesions

In all of the reviewed studies, participants with stroke performed significantly worse than participants in control groups on executive functioning tasks. Three low quality studies found evidence for executive dysfunction following striatal ischaemic lesions (Cools, Ivry & D'Esposito, 2006, Eslinger & Grattan, 1993), posterior lesions (Eslinger & Grattan, 1993) and cerebellar lesions (Manes *et al.* 2009) compared to frontal lesions. Five higher quality studies also examined executive dysfunction in different types of stroke.

Leskela et al. (1999) compared participants with frontal lesions to participants with non frontal lesions using a wide range of executive functioning measures (Trail Making, Verbal

Fluency, Stroop Test and WCST) and found that those with frontal lesions were significantly slower on processing speed and executive functions than those in the control group and were significantly slower on the Trail Making part A and Stroop Test than those with non frontal lesions. They concluded that there was no evidence of dysexecutive syndrome being related specifically to frontal lobe lesions as there was executive dysfunction in both groups as compared to the control group, however, there was some evidence that processing speed may show greater impairments in frontal lesions. This study used a wider range of measures than lower quality studies comparing individuals with frontal and non frontal lesions and had a larger sample size in all of the groups; therefore, these conclusions are likely to carry greater weight and are more convincing evidence of executive dysfunction being related to lesions in areas other than the frontal lobes.

Nys et al. (2006) compared frontal lesions and basal ganglia lesions and found both groups were impaired on executive functioning tasks compared to a control group but that basal ganglia stroke was related to perseveration suggesting that executive functioning may be differentially impaired in frontal compared to non frontal lesions. Only two tests of executive functions were used and the sample sizes in each group were unequal, however, the results suggest that attention and verbal fluency are affected in both frontal and basal ganglia stroke. Rao et al. (1999) carried out a good quality study comparing individuals with anterior circulation stroke to individuals with Transient Ischaemic Attack (TIA), Peripheral Vascular Disease (PVD) and healthy controls and found that those with stroke were more impaired in initiation, mental flexibility and set shifting, however, this conclusion applies only to frontal lesions. The groups each contained a small number of participants which may have limited the findings somewhat.

Su et al. (2007) compared a group of individuals with basal ganglia haemorrhage to healthy controls on the WCST and found that the individuals in the stroke group were significantly poorer than those in the control group; however, conclusions can only be drawn with regard to performance on one test which may not apply to other domains of executive functioning. This study also only assessed those with haemorrhage rather than ischaemic stroke, therefore the results may not generalise to those with ischaemic lesions in the basal ganglia. The overall sample in both the group of individuals with stroke and the control group were younger (mean 53.8 and 56.9 respectively), however, this may be due to the fact that haemorrhagic stroke occurs more frequently in younger adults than ischaemic stroke due to the different aetiology.

Three studies addressed differences between left hemisphere and right hemisphere lesions. Nys et al. (2006) found that the hemisphere of the lesion was not related to perseveration and Planton et al. (2012) found that executive dysfunction was not related to hemisphere of stroke; however, Su et al. (2007) found that those with right hemisphere stroke were significantly poorer than left hemisphere stroke on the WCST. This evidence appears inconclusive, however, Nys et al. (2006) and Planton et al. (2012) had larger sample sizes therefore it is likely that they would have had sufficient power to detect an effect if it had been present. Planton et al. (2012) used the Modified Card Sorting Test and seven other measures of executive functioning and therefore may be able to draw stronger conclusions than the Su et al. (2007) study which used only one measure. It may be that basal ganglia haemorrhage leads to a difference in performance related to hemisphere; however this is unclear from the evidence presented.

These results suggest that both frontal and non frontal areas are involved in executive dysfunction following stroke and supports the theory that there are multiple and bilateral components involved in executive functioning. It does not appear from the studies involved in the review that there is strong evidence for involvement of a particular hemisphere in executive dysfunction following stroke.

Discussion

Fourteen published studies were reviewed in which executive functions were explored following stroke. Previous research has found that 78% of individuals have impairment in at least one cognitive domain following stroke (Lesniack, Bak, Czepiel, Seniow & Człankowska, 2008) and executive dysfunction has been found to occur in 18% to 32% of individuals following stroke (Lesniack et al. 2008; Rasquin et al. 2004).

The results of this systematic review indicate that executive functions are consistently impaired in survivors of stroke across different types of stroke. Historically, executive functioning deficits have been presumed to arise solely from frontal lobe damage (Hodges, 1994) and indeed, several of the included studies observed executive dysfunction amongst stroke survivors with frontal lesions (Rao et al. 1999; Nys et al. 2006, and Leskela et al. 1999). However, advances in neuroimaging have shown that there are multiple connections between the frontal lobes and other cortical, subcortical and brain stem areas (Alvarez & Emory, 2006).

The current review found evidence that non frontal lesions are also implicated in executive dysfunction, for example, Leskela et al. (1999) found executive dysfunction in individuals with lesions in both frontal and non frontal areas. Lesions in the basal ganglia were also found to be implicated in executive dysfunction in the current review (Nys et al. 2006; Su et al. 2007). This is consistent with a meta-analysis of executive functioning measures and neuroimaging which found that both frontal and non frontal regions of the brain are necessary for intact executive functions (Alvarez & Emory, 2006).

Systematic reviews provide evidence for executive dysfunction in Parkinson's Disease (Kudlicka et al. 2011), post traumatic stress disorder (Polak, Witteveen, Reitsma & Olf, 2012), heavy alcohol use (Montgomery, Fisk, Murphy, Ryland & Hilton, 2012) and schizophrenia (Minzenberg, Laird, Thelen, Carter & Glahn, 2009) which provides further support for the involvement of multiple areas of the brain in executive functioning.

Clinical Implications

The National Clinical guidelines for stroke (Intercollegiate Stroke Working Party, 2008) recommend detailed cognitive assessment following stroke in order to quantify the extent and nature of impairments and abilities. HersHKovitz and Brill (2007) report that an

evaluation of cognition can assist the multidisciplinary team to develop and monitor rehabilitation programs and the National Clinical guidelines report that this can also assist with resource allocation (Intercollegiate Stroke Working Party, 2008). With regard to executive functions, the guidelines recommend a comprehensive assessment of executive functioning for individuals experiencing difficulties with concentration due to the impact of this on other cognitive domains and engagement in rehabilitation (Intercollegiate Stroke Working Party, 2008). It is recommended that a detailed assessment of executive functions should be used to plan rehabilitation and guide discussion with the individual, their family and staff supporting them on expectations and consequences.

Early detection of executive dysfunction is important for planning appropriate cognitive rehabilitation strategies, however, current screening measures of cognition may not be sensitive to executive dysfunction (Lesniack et al. 2008), for example, the commonly used Mini Mental Status Examination (MMSE). This is particularly important with regards to executive functions as these types of difficulties have more profound effects on adjustment than other cognitive impairments (Crawford & Henry, 2005). The results of the current review suggest more detailed screening of executive functioning should be carried out following stroke given the evidence that executive functions are commonly impaired following stroke. It also implicates lesions in areas of the brain other than the frontal lobes which provides evidence that screening for executive dysfunction should be carried out regardless of lesion location.

A recent systematic review found evidence that cognitive rehabilitation techniques can improve some domains of executive function following stroke (Poulin et al. 2012). This highlights the clinical importance of screening for impairments to assist with quantifying impairments and developing individually tailored rehabilitation goals and plans.

Executive dysfunction has been found to be a significant predictor of poor functional outcome one year post stroke (Lesniack et al. 2008) and has been implicated in difficulties returning to work (Alvarez & Emory, 2006) which has cost implications to society. It is therefore important to assess for executive dysfunction and develop a rehabilitation plan in order to ensure the best possible functional and occupational outcomes. A comprehensive assessment of executive functions may be more appropriate than single measures due to the range of executive functions that have been shown to be impaired in this systematic review. Impairments in executive functions may also lead to poor performance in other cognitive

domains (Lezak et al. 2004). For example, impairment on memory tasks may be due to difficulties with initiation, perseveration, working memory and retrieval strategies (Crawford & Henry, 2005). It is therefore important to differentiate between the underlying causes of such difficulties in order to optimise rehabilitation efforts and plan effective and appropriate rehabilitation.

Methodological limitations

There are a number of limitations of the current review which may affect the conclusions drawn. Quality ratings were carried out by one individual; therefore there is no inter-rater agreement for the quality of each included study. It would have been beneficial to have a second individual to rate the papers and find out the percentage agreement in ratings. This would improve the reliability of the ratings for each paper. The study may also be vulnerable to publication bias as only published studies were included in the current review. It may have been beneficial to include unpublished data in order to have a greater range of information regarding impairments in executive functions following stroke.

Conclusion

The available evidence suggests that aspects of executive functions are impaired following stroke. There are a number of valid and reliable standardised measures available for assessing different areas of executive functioning, however, a consistent battery is not used across different studies making comparisons difficult and limits the ability to form firm conclusions regarding which areas of executive functioning are consistently impaired following stroke. From the available evidence, processing speed, working memory, attention and cognitive flexibility are the most commonly reported impairments in executive functioning following stroke; however, there is also evidence for impairments in initiation, set shifting, fluency and real life problem solving.

From the studies included in the review, it is clear that areas other than the frontal lobes are implicated in executive dysfunction following stroke. This fits with current theory that executive functioning involves many areas of the brain and multiple systems are involved. There is evidence for impairments following basal ganglia and cerebellar stroke. This suggests that frontal and non frontal areas are involved in executive functioning; however, damage to these areas may result in different patterns of impairment related to

interconnections between these areas. There was no consistent evidence from the current review that hemisphere of stroke was related to executive dysfunction.

Future research should use larger samples and test batteries that sample the variety of different abilities involved in executive functions and that have good evidence of reliability and validity to ensure all areas of executive functioning are assessed. Specific patterns of executive dysfunction should be investigated in different types of lesions and it may be beneficial to further explore whether hemisphere of stroke is associated with executive dysfunction as there is no consistent evidence for this from this review. Given that previous research has found that dysexecutive syndrome can have emotional and social consequences, and that depression and emotional lability are known consequences of stroke (Lincoln et al. 2012), it would be beneficial for future research to address the psychosocial correlates of executive dysfunction following stroke. This research is important as it may contribute to rehabilitation in terms of raising awareness, increasing identification of executive dysfunction and educating professionals, families and carers involved with an individual on how these difficulties may affect other aspects of cognitive, emotional and social functioning. This could also aid the development of compensatory strategies specific to nature of the deficits.

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Part II
Empirical study

2.1 Link between systematic review and empirical study

The systematic review in chapter one highlights that executive functions are consistently impaired following stroke and that this impairment is not limited to frontal lobe stroke. There has been considerable research into the neuropsychological consequences of stroke, (e.g. Lesniak *et al.* 2008) and into the experience of distress (Kneebone & Lincoln, 2012) following stroke. Post stroke depression and emotional lability are the most extensively studied emotional difficulties following stroke (e.g. Hackett *et al.* 2005). Emotion regulation has been investigated in both physical and mental health; however, there is a paucity of research in emotion regulation with survivors of stroke. As a number of emotion regulatory strategies are thought to be risk or protective factors for psychological and physical difficulties (Aldao *et al.* 2010), the concept is worthy of study following stroke given the known physical and psychological difficulties associated with stroke. This chapter will focus on stroke and emotion regulation before outlining the aims and hypotheses. In order to avoid repetition, the information regarding the links between emotion regulation and executive functioning, will be discussed in the journal article.

2.2: Stroke

What is a stroke?

A stroke occurs when the blood supply to the brain is interrupted, leading to neuronal death (Miller, 1994). This can occur through a blockage in the artery due to plaque deposits, blood clots, fat globules or a trapped air bubble (ischaemic stroke) or through a ruptured blood vessel (haemorrhagic stroke). A haemorrhage can occur due to high blood pressure weakening the vessel walls or through aneurysms or arteriovenous malformations (Lincoln *et al.* 2012).

Blood vessels supply the brain with oxygen, glucose and other nutrients (Lezak *et al.* 2004) therefore; disruption to blood flow can lead to irreversible damage to the areas of the brain supplied by the blood vessel in which the stroke occurs (Lincoln *et al.* 2012). The cells surrounding the area of infarction are also at risk due to neurochemical changes following stroke and if treatment is not carried out within a specific time period these cells may be incorporated into the area of infarction (Lezak *et al.* 2004).

The British Heart Foundation statistics database reports that 140,307 individuals in the UK had a stroke in 2009 across all age groups and, although stroke occurs predominantly in those aged over 65 (Stroke Association, 2012), 51,964 of these occurred in those aged under 75 (Scarborough *et al.* 2010). As the proportion of the population over 65 continues to increase, it is likely there will be an increasing number of survivors of stroke (Stroke Association, 2012) and these individuals will have rehabilitation needs. This costs the UK an estimated £8.2 billion each year (Stroke Association, 2012) which has significant implications for the NHS.

Psychological consequences of stroke

There are a number of emotional consequences of stroke, some are considered to be reactive and include anxiety regarding fear of death and fear of falling. A longitudinal study found a prevalence rate of between 22-25% for post stroke anxiety (De wit *et al.* 2008). The most commonly investigated emotional difficulty following stroke is depression and a systematic review found a prevalence of 35% for post stroke depression (Hackett *et al.* 2005).

Other difficulties are thought to be related to possible brain damage such as emotional lability (Lincoln *et al.* 2012) defined as uncontrollable laughing or crying out of context to the situation (Lincoln *et al.* 2012). This has been extensively studied in survivors of stroke with prevalence ranging from 15% (House *et al.* 1989) to 34% (Kim & Choi-Kwan, 2000). There is little investigation into difficulties in emotion regulation post stroke.

2.3 Emotion Regulation

Gross and Thomson (2007) propose that, in order to understand emotion regulation, it is important to understand what is being regulated. Basic emotions are thought to be universal and innate and theory suggests there are discrete categories of basic emotions which more complex emotions are derived from (Power, 2006). The emotion generative process begins with an internal or external stimulus that is attended to and appraised as relevant to an individual's goals (Gross, 1998). This triggers physiological, behavioural and experiential response tendencies (Lang, 1995). Although it is argued that emotions arise through appraisals, Power and Dalgleish (1999) argue emotions can arise in the absence of prior thoughts and can be elicited automatically, for example, in fear conditioning. This may be important in individuals with stroke as difficulties such as emotional lability may not be

adaptive emotional responses and lack of insight may lead to difficulties with awareness of emotions.

Functional accounts of emotions propose that emotions facilitate decision making, prepare the motor response and assist with social interactions (Keltner & Gross, 1999) and are best defined as being short term and biologically based reactions to challenges and opportunities that involve experience, perception, physiology and communication (Keltner & Gross, 1999).

What is Emotion Regulation?

Emotion Regulation refers to a set of 'processes by which individuals influence which emotions they have, when they have them and how they experience and express these emotions' (Gross, 1998, p275). Current theory is that evaluation of emotions and their consequences is important as well as the efforts to adjust the degree of emotional response (Gratz & Roemer, 2004). Although down regulation of negative emotion is a common goal (Gratz & Tull, 2010), emotion regulation also includes increasing, decreasing and maintaining both positive and negative emotion and involves awareness, understanding and acceptance of emotions (Gratz & Tull, 2010). The ability to experience negative emotions yet engage in goal directed behaviour and willingness to experience and accept emotions is also considered important (Gratz & Tull, 2010). Gross (2007) describes emotion regulation as involving the ability to understand and monitor emotions in order to influence the strength with which they are experienced and expressed in order to achieve desired emotional states and goals.

Models and strategies of emotion regulation

Gross' (2007) process model of emotion regulation covers different families of emotion regulatory strategies at multiple points in the emotion generative process. The processes in the model are ongoing and each step influences subsequent steps with ongoing regulation dependant on feedback from the emotional response itself (Richards & Gross, 2000). Antecedent (a priori) and response focused (a posteriori) strategies occur at different points in the emotion generative process. Antecedent focused strategies occur before an emotion is fully generated and can change the trajectory of emotional experience (Gross, 2007), for example, avoidance of a situation known to trigger anxiety. Response focused strategies occur following emotion generation and aim to modulate the experience and expression of emotion, for example drinking alcohol to reduce feelings of anxiety. Although the process

model focuses on strategies; awareness and acceptance of emotions are also important for successful emotion regulation (Gratz & Roemer, 2004). Table 2.1 illustrates the families of emotion regulation occurring at various points in the process.

Table 2.1 Emotion regulation families (Gross, 2007)

Type of regulation	Description
Situation selection (antecedent focused)	Choosing one situation over another due to expectations regarding how desirable the situation is to be in (approach or avoidance).
Situation modification (antecedent focused)	Trying to change the current situation to change its emotional impact - may create a new situation.
Attentional deployment (antecedent focused)	The individual directs their attention in the situation to influence the emotion. Often used when the situation can not be changed, for example, distraction, rumination.
Cognitive change (antecedent focused)	The individual tries to change their appraisal of the meaning of the situation or their capacity to manage the situation particularly with regard to their goals.
Response modulation (response focused)	Attempts to influence the experience of emotion by attempts to change expressive behaviour, experience or physiology, for example, relaxation, alcohol, suppression.

Much of the early research in emotion regulation used students in laboratory settings with explicit instructions to either reappraise (antecedent focused) or suppress (response focused) and did not take individual differences in the use of these strategies into account. Gross and John (2003) developed the emotion regulation questionnaire (ERQ) to measure these individual differences in the strategies of reappraisal and suppression. Using this, Gross and John (2003) found that those who tend to use reappraisal have fewer symptoms of depression, higher life satisfaction and closer relationships. In contrast, those tending to use suppression experienced more negative emotion, lower self esteem and more rumination. Contrary to this, Volokhov and Demaree (2010) found that the ERQ did not predict actual behaviour in an emotion eliciting situation in the laboratory which highlights discrepancies between research in the laboratory and in real life situations.

Gyurak *et al.* (2011) propose that adaptive emotion regulation can be implicit and elicited automatically without monitoring and awareness. Adaptive responses require both implicit

and explicit emotion regulation (Gyurak *et al.* 2011); however, 10% of the general population have poor awareness of emotions (Kimhy, in press) which may impact on explicit emotion regulation. Awareness can be described as the ability to attend to emotions and distinguish between discrete basic emotions using labels (Kimhy, in press). Difficulties with this may impact on successful emotion regulation and have been found to be related to poor social functioning (Kimhy, in press) as emotional expressions may act as social signals and cues during interactions (Gross & John, 2003). Alexithymia is characterised by difficulty with identifying, naming and describing emotions and distinguishing between feelings and bodily sensations of emotion (Mikulajzak & Luminant, 2006). Alexithymia has been found to be a risk factor for physical and psychological health difficulties and it has been debated as to whether it is a stable personality trait predisposing individuals to risk of developing mental health difficulties or if it is a coping mechanism in response to distress (Mikulajzak & Luminant, 2006). There is evidence that alexithymia scores remain stable over time even with increasing distress which indicates it may be a stable personality trait (Mikulajzak & Luminant, 2006).

As alexithymia is associated with difficulties in emotion recognition and regulation (Swart *et al.* 2009), it may be that there is some overlap between the constructs of emotion regulation and alexithymia. The Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) explores concepts other than regulatory strategies such as awareness of emotions and individuals with alexithymia have been found to exhibit a profile with specific difficulties in non acceptance of emotions and lack of emotional clarity (Pandey *et al.* 2008). This is interesting with regard to individuals with stroke, as lack of insight can be a presenting problem (Lincoln *et al.* 2012) which may have implications for awareness and acceptance of emotions which in turn is likely to impact on successful emotion regulation.

Heidemeier and Goritz (2013) found perceived control to be conducive to effective self regulation; however, it may predict more negative reactions to stressful life events. A meta analysis found a greater external locus of control is related to depression in both clinical and control groups (Benassi, Sweeney and Dufour, 1988) and psychological distress is associated with general beliefs regarding lack of control over events (Cheng *et al.* 2013). This is important with regard to stroke due to stroke being a sudden and unexpected life event. Building a greater internal locus of control is a target for intervention with individuals with difficulties in emotion regulation as those with an internal locus of control are better able to regulate their emotions (Blum *et al.* 2002). This is due to a greater belief in their ability to

control their emotional states and reactions (Blum *et al.* 2002) which may be important with regard to stroke as the unexpected event and consequent emotional lability may test the belief regarding their ability to control their emotions. Those with an external locus of control may perceive themselves as having little control over their recovery resulting in increased feelings of hopelessness (Thomas & Lincoln 2006) and reduced participation in rehabilitation.

Emotion regulation in clinical groups

Context is important in defining whether an emotion regulatory strategy is adaptive and emotion dysregulation may actually be an adaptive response in certain situations (Gross, 2007). The relationship between emotion regulation and psychopathology is thought to vary across strategies. In a meta-analysis, problem solving and reappraisal were found to be significantly negatively associated with depression, anxiety and eating disorders and avoidance, rumination and suppression were significantly positively associated with depression, anxiety and eating disorders (Aldao *et al.* 2010) implying that reappraisal is associated with better psychological outcomes.

In line with this, Cameron and Jago (2008) propose that interventions to promote adjustment to physical health conditions should be tailored to address avoidance and suppression and promote reappraisal and proactive behaviours. A number of therapeutic approaches use emotion regulation training (Aldao *et al.* 2010), for example, in Acceptance and Commitment Therapy (ACT), experiential avoidance is thought to maintain psychological difficulties and suppression can increase access to unwanted thoughts (Aldao *et al.* 2010). ACT promotes acceptance which is thought to be important for adaptive emotion regulation (Gratz & Tull, 2010). Cognitive Behaviour Therapy (CBT) utilises problem solving and reappraisal which are also thought to be adaptive emotion regulation strategies. These approaches may be useful with individuals or populations known to experience difficulties with emotion regulation.

2.4 Current study

The systematic review in chapter one highlights that executive dysfunction is common following stroke. The particular aspects of executive functioning found to be impaired were processing speed, working memory, attention and cognitive flexibility which have previously been linked with emotion regulation (Richards & Gross, 2007; Schmeichel &

Demaree, 2010; Gyurak *et al.* 2009). Given that a number of constructs underlying executive functions are also implicated in successful emotion regulation, this makes the relationship between emotion regulation and executive functions particularly interesting in the field of stroke which has both neuropsychological and emotional consequences. The brain areas involved in emotion generation and regulation are closely related to those involved in executive functioning, for example, both involve areas of the prefrontal cortex and subcortical circuitry. Previous research has found that individuals with post stroke emotional lability are more likely to have lesions in frontal and basal ganglia regions of the brain (Tang *et al.* 2009) which, as evidenced in the systematic review, are areas with links to executive functions. Those with post stroke emotional lability have also been found to have poorer executive functioning (Tang *et al.* 2009), therefore, it is possible that a similar pattern will be present for emotion regulation.

Emotion regulation has also been studied extensively in physical health conditions and deficits have been found to negatively impact upon adjustment. As stroke is a physical health condition it is possible that emotion regulation difficulties may be involved in adjustment difficulties following stroke.

Psychological difficulties such as depression and anxiety have also been found to be associated with emotion regulation difficulties including increased use of suppression as a strategy and low levels of acceptance. Depression vulnerability has also been found to be related to deficits in emotion regulation (Ehring *et al.* 2008). As depression and anxiety are commonly found post stroke, these difficulties may be associated with emotion regulation difficulties. The previous chapter reviewed the literature for executive functioning difficulties post stroke and found consistent evidence for executive dysfunction, not limited to frontal lobe lesions. Given the evidence for the structures involved in emotion regulation, it is possible that there is a link between difficulties in emotion regulation and executive dysfunction.

2.5 Aims and hypotheses

The aims of the current study are to explore emotion regulation in survivors of stroke as compared to a control group. Secondary aims were to examine the relationship between emotion regulation, executive functioning and quality of life.

There were six main hypotheses as stated below:

1. Participants with stroke will have significantly more difficulties with emotion regulation than control participants.
2. Participants with stroke will have significantly lower self-reported quality of life than control participants.
3. There will be a correlation between executive dysfunction and greater difficulties in emotion regulation amongst stroke participants.
4. There will be a correlation between greater difficulties in emotion regulation and lower self reported quality of life amongst stroke participants.
5. There will be a correlation between executive dysfunction and lower self reported quality of life amongst stroke participants
6. There will be a correlation between greater difficulties in emotion regulation and higher self reported anxiety and depression amongst stroke participants.

Chapter 3
Extended methodology

This chapter details the methodology employed in the study including design, participants, ethical considerations, measures used, procedure and data analysis.

3.1 Design

The study involved a cross sectional design. For hypotheses one and two, participants with stroke were compared to a control group without stroke on the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004), Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) and the World Health Organization brief quality of life measure (WHOQOL-BREF; Harper & Power, 1998). Each participant was seen individually on one occasion. For hypotheses three to six, a within subjects design was employed in order to find out if there were correlations between measures used within the group of individuals with stroke.

3.2 Participants

This section details the methods used to recruit participants in each group. It also outlines the inclusion and exclusion criteria adopted for each group in the study.

3.2.1 Inclusion and exclusion criteria for stroke participants

Inclusion criteria

- Diagnosis of stroke (both ischaemic and haemorrhagic)
- Medically stable (not currently an inpatient)
- Able to give informed consent
- Age over 18 years old

Exclusion criteria

- Current or previous medical history known to affect cognitive function e.g. dementia, head injury involving loss of consciousness, alcohol abuse, degenerative condition, or learning disability.
- Visual or hearing impairments that would impact on their ability to complete neuropsychological assessments or questionnaires that cannot be rectified using visual or hearing aids.

- Currently aphasic or suffering from receptive aphasia
- Transient ischaemic attacks rather than stroke diagnosis

3.2.2 Inclusion and exclusion criteria for control participants

Inclusion criteria

- These were the same as the stroke participants other than the control group did not have a diagnosis of stroke.

Exclusion criteria

- These were the same as for the stroke participants.

Potential control participants were provided with the same information regarding the study as stroke participants, however, the information sheet for the control group was changed to explain why they had been chosen as a control participant and what the study would entail for them should they choose to take part.

3.2.3 Demographic information collected

The following information was recorded from all participants

- Gender
- Age
- Years of formal education
- Current/previous occupation

The following information was recorded for stroke participants only

- Type of stroke (Oxford classification)
- Location of lesion
- Time since stroke

3.2.4 Recruitment

Potential stroke participants were identified by the NHS Grampian Stroke Audit Coordinator from the NHS Grampian stroke register. The stroke register was viewed in February and March 2012 for participants who received a stroke diagnosis between November 2010 and February 2012 inclusive. A total of 180 individuals with a diagnosis of stroke were identified as eligible to participate in the study through applying the inclusion and exclusion criteria. All 180 eligible participants were sent a letter outlining the purpose of the study

(appendix six) along with an information sheet (appendix seven) with details regarding participation. A form requesting contact details was also sent with the letter as well as a stamped, addressed envelope to enable them to return the form. The chief investigator telephoned those who returned their forms expressing an interest in participating in the study. An appointment for a ninety minute assessment was then made. Six participants returned their contact sheets and agreed an appointment date but did not attend the appointment. Four returned their contact sheets but when telephoned, decided not to take part in the study. A total of 50 individuals with stroke agreed to take part in the study and attended an appointment where they were provided with a consent form (appendix nine) and given the opportunity to ask questions.

Control participants were recruited through a local community group (local church). Every effort was made to match individuals in the control group with those in the stroke group in terms of age, gender and years of education. They were approached as a group in the church hall after the church service and were provided with an information sheet (appendix eight) and a consent form (appendix ten). They were given the option to complete the study after the church service or to sign up for a home visit. No incentive was offered for their participation in the study. A total of 45 control participants agreed to take part in the research.

3.3 Ethical Considerations

The following ethical issues were considered:

- Potential distress to participants
- Informed consent
- Confidentiality

3.3.1 Potential distress to participants

Some of the questionnaires asked questions about mood, anxiety and emotional state. One of the questionnaires asked how participants dealt with strong emotions. It was acknowledged that this may cause distress; therefore, participants were informed that they did not have to complete the questionnaires if they found them upsetting. The neuropsychological assessments were identified as having the potential to lead to fatigue or distress for stroke participants, therefore, it was planned to discontinue testing should this happen. Participants were made aware that they could stop testing for breaks or could

complete the assessments over two appointments. All participants were also informed that they could withdraw from the study at any time without having to give a reason and that this would not affect their medical care in any way.

3.3.2 Informed consent

Participants were provided with an information sheet detailing why they had been asked to participate in the study and what participation would involve. Participants were given the opportunity to ask questions and discuss the study with the chief investigator before deciding whether or not to take part in the research.

3.3.3 Confidentiality

Each participant was allocated a number for identification purposes but all data was anonymised and kept separately from any identifiable data. Participant consent forms and contact sheets were kept separately from study data. Data with personally identifiable information was stored in a locked cabinet in an NHS building and was destroyed at the end of the study.

Ethical approval for the study was granted by the North of Scotland Research Ethics committee on 14th December 2011 (REC number 11/NS/0041; appendix four). Approval from the Research and Development department of NHS Grampian was granted on the 16th January 2012 (appendix five).

3.4 Measures

Participants with stroke completed the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004), Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983), Verbal Fluency task, Brixton Spatial Anticipation Test, Hayling Sentence Completion Test, Color Trails Test, WHOQoL-BREF and National Adult Reading Test (NART). Demographic information was also collected. The ordering of the neuropsychological tests was counterbalanced to account for effects of the tests on each other and participant fatigue. In total, participants with stroke completed eight measures.

Participants in the control group completed the HADS, DERS, WHOQoL-BREF and NART. Demographic information was also collected. In total, control participants completed four measures.

3.4.1 Validated questionnaires

Three self report measures were completed by all participants. These were the Hospital Anxiety and Depression Scale, the Difficulties in Emotion Regulation Scale and the World Health Organization brief quality of life measure.

Hospital anxiety and depression scale (HADS; Zigmond & Snaith, 1983)

The HADS is a brief measure of anxiety and depression used descriptively in the current study to compare the participants in the stroke group to those in the control group. It is a 14 item self report measure developed to detect anxiety and depression in medical outpatient settings where seven items relate to depression and seven to anxiety (Zigmond & Snaith, 1983). Each item on both the anxiety and depression subscales is given a score between zero and three and this is summed to give a total score for each subscale. In the current study, the means for each group were compared to look for significant differences and each subscale was used in the correlational analyses. The numbers of participants falling into each category (Snaith & Zigmond, 1994) of 'mild' (8-10), 'moderate' (11-15) or 'severe' (16 and above) was calculated for each group. Cut off scores are available for stroke patients (Bennett & Lincoln, 2004), however, these range from five (Johnson *et al.* 1995) to eight (Aben *et al.* 2002) for mild depression and six (Johnson *et al.* 1995) to seven (Aben *et al.* 2002) for mild anxiety and there is no consensus across studies as to what this should be. These studies had differing sensitivity and specificity with Johnson *et al.* (1995) presenting good sensitivity but low specificity. In a review of 747 studies using the HADS in psychiatric, somatic, primary care and general populations, Bjelland *et al.* (2002) found that using eight as the cut off achieved the optimal balance between sensitivity and specificity. Aben *et al.* (2002) found the accepted cut off scales for the HADS depression and anxiety subscales as specified by Snaith and Zigmond (1994) were optimal in a stroke population. For this reason, the original cut off scores were used to compare groups in the current study which is consistent with Bennett *et al.* (2006) where a score of greater than seven on these subscales was used to classify individuals with stroke as having mild depression or anxiety.

The HADS has been recommended for screening of mood in stroke patients without communication problems (Bennet & Lincoln, 2004) as it is relatively short and has been suggested to be easier for stroke patients to complete than other measures of anxiety and depression (O'Rourke *et al.* 1998). It has been described as placing low demands on memory (Bennett & Lincoln, 2004) which is important in acquired brain injury. The HADS does not rely on physical or somatic items and so is less likely to be affected by misleadingly high

scores due to non-anxiety or depression related somatic symptoms in a population with physical illness making it a suitable measure for stroke survivors. Crawford, Henry *et al.* (2001) assessed 1792 non clinical participants on the HADS and found a significant, moderate correlation (0.53) between the anxiety and depression subscales. Cronbach's alpha for the anxiety subscale was 0.82 and 0.77 for the depression subscale (Crawford, Henry, *et al.* 2001). This indicates good reliability of the HADS.

Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004)

This measure assesses six domains of emotion regulation and was used in the current study to explore differences in emotion regulation between the participants in the stroke group as compared to those in the control group. It was also used to investigate associations between difficulties in emotion regulation, executive functions and quality of life in the participants in the stroke group. This scale has 36 items which make up six domains of emotion regulation and a total score and has previously been used with a stroke population (Scott, in preparation). In the analysis, groups were compared on mean scores for all six domains of this measure and the total score with higher scores reflecting greater difficulties in emotion regulation. For the correlational analysis, only the total score was used. Evidence for the validity and reliability of the six domains and the total score comes from the initial validation of the measure using undergraduates, 73% female aged 17 to 55. The validity and reliability of the total score and each domain will be discussed below.

- non acceptance of emotional responses (non acceptance)
- difficulties engaging in goal directed behaviour (goals)
- impulse control difficulties (impulsivity)
- lack of emotional awareness (awareness)
- limited access to emotion regulation strategies (strategies)
- lack of emotional clarity (clarity)

The total score was reported to have high internal consistency (Cronbach's alpha = 0.93) and test-retest reliability of 0.88 (Gratz & Roemer, 2004). For the non acceptance subscale, internal consistency was 0.85 and test-retest reliability was 0.69. Goals had an internal consistency of 0.89 and test-retest reliability of 0.69, impulsivity had internal consistency of 0.86 and test-retest reliability of 0.57. The awareness subscale had internal consistency of 0.8 and test-retest reliability of 0.69, the strategies subscale had internal consistency of 0.88 and test-retest reliability of 0.89 and finally, the clarity subscale had internal consistency of

0.84 and test retest reliability of 0.8. To establish construct validity, the original validation study carried out correlations with the Negative Mood Regulation Scale (NMR) and found that all DERS subscales were positively correlated with the NMR (Gratz & Roemer, 2004).

Participants were asked to read each statement and rate how often it applied to them on a scale of one to five with one being 'almost never' through to five being 'almost always' (Gratz & Roemer, 2004). The DERS has been used in a number of different populations and found to be significantly associated with deliberate self harm, binge eating, generalised anxiety, partner abuse in men and cocaine use which are all thought to be forms of regulating emotions (Gratz & Tull, 2010). It has also been found to be associated with mood difficulties including depression and anxiety severity and experiential avoidance (Gratz & Tull, 2010). It has been used with individuals with eating disorders (Harrison *et al.* 2009, 2010), depression (Ehring *et al.* 2008), generalised anxiety disorder (Salter-Pedneault *et al.* 2006, Staples & Muhlman, 2012) post traumatic stress disorder (Tull *et al.* 2007), psychogenic non epileptic seizures (Uliaszek *et al.* 2012), stroke (Scott, in preparation) and has been found to be sensitive to change following intervention for cocaine use (Fox *et al.* 2007).

Further evidence for the validity of the DERS comes from a study by Staples and Muhlman (2012) examining the psychometrics of the DERS in a sample aged 60 to 88 with generalised anxiety disorder (GAD) compared to healthy controls. It was found that the DERS was correlated with the anxiety sensitivity questionnaire, the Penn State Worry Questionnaire, the Beck Depression Inventory and the GAD questionnaire for the DSM-IV. The two groups were significantly different on the subscales of the DERS with average total score differing by 20 points (Staples & Muhlman, 2012). Good internal consistency was found for the full sample, the individuals with GAD and the control group (all $\alpha > 0.85$). There was divergent validity with all measures of anxiety and depression and test retest reliability was 0.53 (Staples & Muhlman, 2012). Overall, lower DERS scores were noted in this age group as compared to previous research in GAD in younger adults, however, the evidence shows that the DERS can be used with an older adult group in a research setting (Staples & Muhlman, 2012). This is important for the current study given that the likelihood of stroke increases with age, leading to the likelihood of an older age sample. The DERS was chosen over other measures of emotion regulation as it assesses a wider range of areas thought to be important in successful emotion regulation such as awareness and acceptance of emotional responses rather than comparing strategies.

World Health Organization Quality of Life questionnaire (WHOQoL – BREF)

This measure assesses self-reported quality of life and was used to explore differences between individuals with stroke as compared to those in the control group. It was also used to explore associations between quality of life, executive functions and emotion regulation. This is a 26 item self report questionnaire and asks participants to think of their life in the past four weeks. It was developed as a shorter version of the 100 item World Health Organization Quality of Life measure and derived from the cross cultural data collected during the development of the WHOQOL-100 using 13 pilot areas applying sampling quota of 50% of people under age 45, 50% male and 250 people with disease to 50 well people (Harper & Power, 1998). It assesses the same four domains of physical health, psychological health, social relationships and environment as the WHOQOL-100. Each score indicates perceived levels of quality of life in each domain and higher scores indicate greater quality of life. In the current study, raw scores on the WHOQOL-BREF were added up for each domain and converted to scores between 0-100 using tables provided in the manual (World Health Organization, 1996) to allow comparisons between the domains. These converted scores were used in the analyses. Mean scores for each group were used to compare the individuals with stroke to the control group for each domain.

Correlations between the four domain scores on the WHOQOL-100 and WHOQOL-BREF range from 0.89 to 0.95 and the internal consistency of the four domains on the WHOQOL-BREF ranged from 0.66 to 0.84 (Harper & Power, 1998). Test retest reliability on the WHOQOL-BREF is 0.66 for physical health, 0.72 for psychological health, 0.76 for social relationships and 0.87 for environment (Harper & Power, 1998). A field trial was carried out to examine the properties of the WHOQOL-BREF (Skevington *et al.* 2004) where data was gathered from 24 areas in different countries from 11830 respondents (53% female) aged 12 – 97 years with a mean age of 45 (Skevington *et al.* 2004). The results were used to establish discriminant validity, construct validity and internal consistency. For physical health, psychological health and environment, Cronbachs alpha for internal consistency were 0.82, 0.81 and 0.80 respectively, however, for social relationships was 0.62 (Skevington *et al.* 2004). This is evidence that the WHOQOL-BREF is an appropriate brief substitute for the longer WHOQOL-100 which was an important consideration for the population under investigation in the current study.

3.4.2 Neuropsychological measures

Measurement of executive dysfunction has been described as the most problematic area of neuropsychological assessment (Crawford & Henry, 2005) and the approach taken to assessment is the subject of ongoing controversy. At present, there is no gold standard battery for the approach to assessment of executive functioning (Kudlicka *et al.* 2011).

Burgess *et al.* (1998) argue that an assessment of executive functioning should at least include a general measure of inhibitory abilities, executive memory abilities and a measure of multitasking. This can be supplemented by measures assessing rule attainment, planning, initiation and abstract reasoning. In addition to this, Witsken *et al.* (2008) argue that sustained attention, inhibition, set shifting and problem solving should comprise a comprehensive assessment of executive functions.

For the purpose of the current research, four standardised neuropsychological measures were used to assess different aspects of executive functioning. The measures chosen and the concepts they are thought to measure are:

- Verbal fluency - Mental flexibility
- Brixton spatial anticipation test – Rule detection and set shifting
- Hayling sentence completion test - Inhibition
- Color Trails Test interference index – sequencing, selective attention, set shifting

These were completed by the stroke participants only and are described in further detail below. Larger or fixed batteries were considered such as the Behavioural Assessment of the Dysexecutive Syndrome (BADS; Wilson *et al.* 1996) which has high ecological and face validity (Bennett *et al.* 2005), however, there are no norms based on age for the subtests and administration of the entire battery is required for a standardised score (Lincoln *et al.* 2012). A number of investigations have provided mixed evidence for subtests of the battery and a composite profile score may mislead as it does not provide information with regard to the aspects of executive functions that are impaired. Research into the BADS has also found effect sizes for the differences between clinical and non clinical groups were small to moderate and the majority of subtests have low sensitivity and unknown specificity (Crawford & Henry, 2005). Bennett *et al.* (2005) propose that a mixed battery of subtests of executive functions may be more suitable.

For the purposes of the current research, the four measures chosen were thought to cover the above criteria by Burgess *et al.* (1998) and Witsken *et al.* (2008). In addition to this, the Brixton, Hayling and verbal fluency assessments have been found to have normal distributions in non clinical groups as well as moderate ecological validity (Crawford & Henry, 2005; Burgess & Shallice, 1997).

Verbal Fluency Test (FAS – letter fluency and category fluency)

This is an executive functioning test sensitive to disorders of the frontal lobes (Henry & Crawford, 2004) and can be used with individuals from age 20 onwards with English as a first language. In the current study, for letter fluency, the participants were asked to name as many words as possible beginning with a given letter in one minute excluding proper nouns and similar words with a different suffix. This assesses letter (phonemic) fluency. The letters used were F, A and S. The raw score is the total number of correct words across these three trials and this was converted to a t score based on age, gender and years of education using norms from Gladsjo *et al.* (1999). A fourth trial asking individuals to generate as many animal names as possible in one minute was also used to assess category (semantic) fluency. The raw score was the total number of animals named in one minute and this was converted to t scores as above which were then used in the correlational analyses. Higher t scores reflect better performance. The normative sample involved 768 people (52% male) age 20 to 101 years old ($m = 50.4$) with a range of two to 20 years of education ($m = 13.6$) (Gladsjo *et al.* 1999). This assessment was used to measure mental flexibility.

Lezak *et al.* (2004) described this test as a “sensitive indicator of brain dysfunction” (p520) and Crawford & Henry (2004) argue this measure has good norms and reliability as well as moderate ecological validity and moderate sensitivity. A meta analysis by Henry and Crawford (2004) investigated 31 studies including 1791 participants and found “strong support for the validity of both phonemic and semantic fluency as an executive measure” (p294) that may be more sensitive than the Wisconsin Card Sorting Test. Henry and Crawford (2004) found that semantic fluency is sensitive to temporal lobe damage and both phonemic and semantic fluency place comparable demands on the frontal lobes. This provides evidence for its suitability in the current study as a measure of executive dysfunction.

Brixton Spatial Anticipation Test (Burgess & Shallice, 1997)

The Brixton Test (Burgess and Shallice, 1997) is a rule detection and following task designed to assess executive functions. In addition, the task also assesses response flexibility to the changing rules and the executive component of working memory (Burgess and Shallice, 1997). Impaired performance has been found in those with disorders thought to involve executive processes (Strauss *et al.* 2006). This task uses an A4, 56 page book with each page containing two rows of five circles numbered one to ten. One of the circles is blue and the location of the blue circle changes as the pages are turned according to a particular pattern which the individual has to work out. There is no time limit and participants are not informed whether their response is correct. This requires the participant to detect and follow rules and then shift set and detect the new rule. The raw score is the number of errors made and this is converted to a Sten score. Crawford and Henry (2005) report that Sten scores are coarsely grained and may obscure differences in raw scores, therefore, for the purpose of the current research, the number of errors made on the Brixton was used in the analyses. In the analysis, a higher score reflects poorer performance as it is number of errors rather than the sten score. There are significant correlations between performance on the Brixton and premorbid IQ (Burgess & Shallice, 1997), therefore, years of education was controlled for in the analysis.

This assessment is conceptually similar to the Wisconsin Card Sorting Test which is a well known assessment of rule attainment and set shifting, however, Burgess and Shallice (1997) argue that the Brixton assessment is straightforward and less stressful for participants. Burgess and Shallice (1997) report that the difficulties assessed by the Brixton are among the most commonly demonstrated impairments in people with executive dysfunction.

Crawford and Henry (2005) argue that this is a good assessment of executive dysfunction as it is derived from theory and has moderate reliability, specificity and sensitivity. The Brixton test was standardised on individuals aged 18 to 80 with anterior lesions, posterior lesions, bifrontal lesions and a control group (Burgess & Shallice, 1997). For the control group, the split half reliability was 0.62 and test retest reliability was 0.71 (Burgess & Shallice, 1997). Reverberi *et al.* (2005) administered this test to 40 individuals with focal frontal lobe lesions (vascular, tumour and traumatic brain injury) and 43 controls and found that all patients were impaired as compared to controls. This suggests it is a sensitive measure of brain dysfunction.

Hayling Sentence Completion Test (Burgess & Shallice, 1997)

This was used to measure initiation and inhibition and is associated with dysfunction in everyday life (Burgess & Shallice 1997). This assessment was derived from theory based on the multi process Supervisory Attentional System (Burgess & Alderman, 2004). Individuals are given two sets of fifteen sentences which have the last word missing. In part one, individuals are asked to complete the sentence as quickly as possible, for example, '*the old house will be torn....*'. In this example, the word '*down*' could appropriately complete the sentence. There are raw scores for time taken and number of errors which are converted to scaled scores. In part two, individuals are asked to complete the sentence as quickly as possible with a word that is unconnected to the meaning of the sentence. This involves inhibition of the correct response and generation of a new response for example, '*most cats see very well at....*'. The word '*night*' would be inappropriate to complete this; however, '*banana*' could appropriately complete this sentence as the word must be completely unrelated to the context of the sentence. There are raw scores for time taken and for number of errors which are divided into types of errors. This is then converted into a scaled score. Scaled scores from parts one and two are summed and converted to a total scaled score. For the purposes of the current research, the total scaled score was used as a measure of inhibition. Higher scores therefore reflect better performance. Performance on the Hayling has been found to be associated with IQ (Burgess & Shallice, 1997), therefore, years of education was controlled for in the analysis.

The standardisation sample consisted of 91 individuals with anterior, posterior and bifrontal lesions and a control group of 121 individuals aged 18 to 80 (Burgess & Shallice, 1997). Patients with frontal lobe lesions have been shown to have double dissociations on performance on the Hayling and Brixton tests (Burgess & Shallice, 1997) showing that they measure different areas of executive functioning. Vascular lesions were present in 39.2% of participants in the standardization sample making it an appropriate measure for this group.

The Hayling has test retest reliability 0.76 for the total scaled score (Burgess & Shallice, 1997). De Frias *et al.* (2006) explored the construct validity of the Hayling, Brixton, Stroop Test and Color Trails part 2 in older adults free from illness. This study found that a single factor accounted for the underlying organization of these tests and that the Hayling loaded with the other tests of executive functioning on the same factor which provides some construct validity.

The Color trails test (D'Elia *et al.*1996)

This test is similar to the Trail Making Test (TMT; Reitan, 1958) but has been adapted by replacing the English alphabet letters with colour. This is argued to minimise the cultural and linguistic bias of the original TMT. The TMT was originally developed as a non language measure of intelligence and was found to be sensitive to brain damage (D'Elia *et al.* 1996). It can be used with individuals aged from 18 to 89 and has norms from 1528 participants based on age and years of education (D'Elia *et al.* 1996).

In Color Trails part one (CT1), individuals are asked to draw a line connecting each circle in ascending numerical order. In Color Trails part two (CT2), individuals are asked to draw a line connecting each circle in ascending numerical order, however, in part two; there are two of each number, one pink and one yellow. Individuals are asked to switch between pink and yellow each time they move up a number in the sequence. For example, one – yellow, two – pink, three – yellow. The time taken to complete is the raw score which can be converted to a t score based on norms for age and years of education. As part one is considered to measure processing speed and part two is considered to have an executive component, it is possible to calculate an 'interference index' (D'Elia *et al.* 1996) by using the formula:

$$\frac{\text{time CT2-time CT1}}{\text{time CT1}}$$

This index is considered a purer measure of selective attention (Strauss *et al.* 2006) controlling for the effect of processing speed. This was the score used for the correlations in the current study. The manual classifies scores on an ordinal scale according to the percentile range they fall into for age and years of education (<1; 2-5; 6-10; 11-16; >16). As other scores in the current study are in the form of continuous variables, raw scores were used for the interference index in the analyses. This provides a greater range of scores for analysis. Years of education was controlled for in the analysis with the interference index as this score does not take years of education into account.

The authors report good temporal stability over a brief period of time (D'Elia *et al.* 1996) and good content validity as it is not as affected by cultural influences as the TMT. The test retest reliability of part one is 0.64 and for part two it is 0.79 (D'Elia *et al.* 1996). The CTT was found to be significantly correlated with the TMT in the normative sample and the color

trials time variables were significantly correlated with each other in normative and clinical samples (D'Elia *et al.* 1996).

National Adult Reading Test (NART; Nelson & Willison, 1991)

This measure was used in the current study as an indicator of premorbid IQ and the number of errors made was used to calculate the predicted full scale IQ using the conversion tables in the manual (Nelson & Willison, 1991). Nelson and Willison (1991) argue that the NART has “obvious potential as a criterion for group matching in research studies” (p7) therefore, it was used to compare the group of participants with stroke to the participants in the control group and to control for differences in premorbid IQ in the analysis.

The NART is composed of 50 words written on a card that are read aloud with no time limit. It is reported that the NART is less stressful than other cognitive assessments and places less demands on attention and concentration (Nelson & Willison 1991) making it a suitable measure for the population in this study. The NART was developed as a premorbid assessment of intelligence (Nelson & Willison 1991) used for determining whether an individual's performance on neuropsychological assessment represents a decline from prior functioning. If a word is pronounced correctly, it is assumed that this is due to prior knowledge of the word (Lezak *et al.* 2004). This allows interpretation of current functioning in the context of premorbid functioning.

Crawford, Parker and Besson (1988) found a split half reliability of 0.9, inter rater reliability between 0.96 and 0.98 and test retest reliability of 0.98 in individuals aged 16 to 88 and O'Carroll (1987) also found high inter rater reliability. Factor analysis of the performance of 139 participants on the NART and the WAIS found that the NART had validity as a general measure of intelligence in adults (Crawford, 1989). Crawford, Deary *et al.* (2001) found the correlation between age 11 IQ and the NART was 0.69, between IQ age 11 and IQ age 77 was 0.64 and between NART and IQ age 77 was 0.63.

Lezak *et al.* (2004) reports that the NART may underestimate premorbid ability if an individual has language difficulties and cautions against its use in those with aphasia, dyslexia or articulatory or visual acuity deficits. Further to the initial standardisation of the NART, evidence has shown that there is some impairment on NART performance in dementia, however, this is reportedly mild in comparison to the effects of dementia on other measures of cognitive function (Lezak *et al.* 2004) and the NART is thought to be resistant

to the effects of age up to 84 years old (Nelson & Willison 1991). O'Carroll (1992) presents evidence that the NART may not hold in certain conditions, for example, Korsakoffs and Morris *et al.* (2005) found a significant correlation of 0.31 between the NART and the Glasgow Coma Scale which is an index of severity of head injury. This indicates that the NART may underestimate premorbid ability with increasing severity of head injury. Given this evidence, scores were interpreted with caution in the current study.

3.5 Statistical power and sample size

Statistical power and sample size was calculated for the different hypotheses separately. Gpower (Faul *et al.* 2007), a power analysis program and Cohen (1992) were used to calculate sample size.

Hypotheses one and two

1. Stroke participants will have significantly more difficulties with emotion regulation than control participants.
2. Stroke patients will have significantly lower self reported quality of life than control participants.

Using Gpower, F Test Family, F test, ANOVA: fixed effects, omnibus, one way and a priori type of power analysis, the sample size required to detect a hypothesised moderate large effect size (effect size $f = 0.3$) at the 0.05 level (one-tailed) with power of 0.8 (high), is 90 participants (45 stroke participants and 45 control participants).

A hypothesised moderate-large effect size was anticipated because Harrison *et al.* (2010) found a large effect size when comparing individuals with depression to individuals without depression using the DERS and Roemer *et al.* (2009) found a large effect size using the DERS to compare individuals with generalised anxiety disorder to a control group. Harrison *et al.* (2009) also found a large effect size comparing individuals with anorexia nervosa to a control group on the DERS. This suggests that clinical groups that are more likely to have mood difficulties are likely to have difficulties with emotion regulation. As the WHOQOL-BREF has been found to have good discriminant validity between individuals who are well and unwell, a moderate-large effect size was anticipated due to the individuals in the stroke sample being compared to a control group without a medical history known to affect cognitive functioning.

Hypothesis three to six

3. There will be a correlation between executive dysfunction and greater difficulties in emotion regulation amongst stroke participants.
4. There will be a correlation between greater difficulties in emotion regulation and lower self reported quality of life amongst stroke participants.
5. There will be a correlation between executive dysfunction and lower self reported quality of life amongst stroke participants.
6. There will be a correlation between greater emotion regulation difficulties and higher self reported anxiety and depression amongst stroke participants.

Using Gpower, Exact test family, correlation: bivariate normal model, one way and a priori type of power analysis the sample size required to detect a hypothesised moderate large correlation (effect size $r = 0.4$; moderate: Cohen & Holiday (1982) at the 0.05 level (one-tailed) with power of 0.8 (high) is 37 participants. This hypothesis relates to the stroke participants only.

3.6 Analysis

Neuropsychological raw test scores for verbal fluency were compared to published normative data for age and years of education and converted to t scores. Scores for the Hayling were converted to scaled scores as advised by the manual (Burgess & Shallice, 1997). The number of errors made on the Brixton was used in the analysis.

Exploratory data analysis

Initially, data was analysed using descriptive statistics and explored using the Statistical Package for Social Sciences (SPSS) version 19. Exploratory data analysis was carried out to check for outliers, normal distribution and to explore differences between the two groups in their age, sex, years of education and NART scores. Analysis of the data for normality, homogeneity of variance, skew and kurtosis was carried out. The Kolmogorov-Smirnov test of normality, Q-Q plots, detrended normal QQ plots, histograms, boxplots, skew and kurtosis were screened to assess the normality of variables (Pallant, 2011). Skew and kurtosis scores were transformed to z scores by dividing the score by the standard error for skew and by square rooting this value for kurtosis (Field, 2005) and were assessed as significant if the score was greater than 2.58 (Clark-Carter, 1997).

Independent samples t tests were carried out to look for differences between the two groups on demographic variables. Chi square was used to look for differences between the groups in gender. When the data did not meet requirements for homogeneity of variance (Levene's test violated) the Welch statistic was reported (Pallant, 2011).

Between group comparisons

Transformation of data was attempted where assumptions for parametric statistics were not met. This occurred in four subscales of the DERS: goals, impulsivity, strategies and clarity. Based on the distributions of the histograms, logarithm transformations were used (Pallant, 2011; Clark-Carter, 1997) but these were unsuccessful and did not improve skew in either the control or stroke group data. Independent samples t tests for parametric data and Mann-Whitney U tests for non parametric data were therefore planned, however, as estimated premorbid IQ (as measured by the NART) and years of education were significantly different between the control and stroke groups, they were explored as covariates. Preliminary checks were conducted to ensure that there was no violation of the assumptions of normality, linearity, homogeneity of variances, and homogeneity of regression slopes (Pallant, 2011). Years of education was chosen as a covariate rather than NART IQ as these variables were strongly correlated with one another (Clark-Carter, 1997) and the NART IQ did not meet the assumption of homogeneity of regression slopes.

Although there is no non parametric equivalent of ANCOVA, parametric tests have been found to be robust when some assumptions are violated (Clark-Carter, 1997). If there is a large sample, particularly over 30 in each group, parametric statistics are more robust to violations of normality (Motulsky, 2010). The non normal data were inspected and it was found that skew was significant, however, kurtosis was not significant for these variables and the histograms did not show bimodal distributions. Vickers (2005) present evidence that ANCOVA is favourable to Mann-Whitney in such cases. This approach is similar to Harrison *et al.* (2010) when dealing with DERS variables therefore, the data for all of the DERS subscales were analysed using ANCOVA.

The physical health domain of the WHOQOL-BREF did not meet the requirements for parametric statistics and based on the histogram distribution a successful square root transformation was carried out. All domains of the WHOQOL-BREF in subsequent analysis with this domain were also transformed in the same way. This is advised by Clark-Carter

(1997) if a statistical test is analysing differences between means. This enabled parametric tests to be used with the WHOQOL-BREF data.

Correlations

Hypotheses three to six involved correlational analyses. Preliminary analyses were carried out to ensure no violations of the assumptions of normality, linearity and homoscedasticity (Pallant, 2011). As multiple comparisons increase the likelihood of a type one error, a conservative p-value of .01 was chosen in order to manage the likelihood of significant results due to chance. This was chosen over the use of a Bonferroni correction as the Bonferroni correction may increase the likelihood of a type two error and mask significant results (Field, 2005). Bivariate one tailed correlations were used to explore statistical associations between performance on executive functioning tasks, emotion regulation and quality of life. The executive functioning measures, DERS total score and three domains of the WHOQOL-BREF were normally distributed enabling parametric statistics to be used, therefore, Pearson product moment correlations were used. Partial correlations were carried out with the Hayling, Brixton and Color Trails interference index in order to control for years of education which is known to affect scores on these assessments. The physical health domain of the WHOQOL-BREF was not normally distributed therefore; correlations with this domain used the non parametric Spearmans rank order correlations in the analysis.

Chapter 4
Journal Article

Emotion regulation, Executive Functioning and quality of life in stroke

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Running Head: Emotion Regulation in stroke

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Emotion regulation, Executive Function and quality of life in stroke

Abstract

Background: Mood disorders, psychological distress and neuropsychological impairments are common consequences of stroke. Both depression and emotional lability have been studied extensively amongst stroke survivors. This study was designed to investigate difficulties in emotion regulation and quality of life after stroke. The relationships between emotion regulation, anxiety and depression, quality of life and executive functioning were also investigated.

Method: Fifty individuals with a stroke and forty-five control participants completed the Hospital Anxiety and Depression Scale, Difficulties in Emotion Regulation Scale, World Health Organization Quality of Life Scale – brief version and the National Adult Reading Test. In addition, individuals with stroke completed four measures of executive functions; Color Trails Test, Verbal Fluency, Brixton Spatial Anticipation Test and the Hayling Sentence Completion Task.

Results: The individuals in the stroke group had significantly greater emotion regulation difficulties and significantly lower self-reported quality of life compared to individuals in the control group. Greater difficulties in emotion regulation were significantly associated with lower self reported quality of life and higher levels of anxiety and depression. No associations were found between difficulties in emotion regulation and executive dysfunction.

Conclusion: Difficulties in emotion regulation were found post stroke as compared to a control group and were associated with higher levels of anxiety and depression and lower quality of life. Future research should explore emotion regulation and its relation to lesion location following stroke and whether there are any links between other aspects of cognition and emotion regulation.

Abstract: 241 words

Introduction

Stroke occurs in approximately 150,000 individuals in the UK each year, predominantly in those aged over 65 (Stroke Association, 2012). As the aging population is increasing, this will lead to a greater number of individuals with stroke with rehabilitation needs. Although physical and cognitive difficulties have been extensively researched and treatment and rehabilitation techniques developed, individuals commonly experience psychological distress and difficulties including depression and emotional lability following stroke. Detection of mood disorders is important following stroke (O'Rourke, MacHale, Signorine & Dennis, 1998) as such disorders may impact on ability to engage in rehabilitation and have implications for well-being and outcome for individuals.

A systematic review of 51 studies found a prevalence of 35% for post stroke depression (Hackett, Yapa, Parag & Anderson, 2005) and one study found that 28% of those not meeting study criteria for depression none-the-less reported cognitions related to worthlessness, hopelessness and suicidality (Hackett, Hill, Hewison, Anderson & House, 2010). This may lead to depression in the longer term. Emotional lability has been defined as uncontrollable laughing or crying disproportionate to the situation (Lincoln, Kneebone, Macniven & Morris, 2012) and studies report differing prevalence rates following stroke. For example, one study reports prevalence as 15%, 21% and 11% at one, six and twelve months respectively (House, Dennis, Molyneux, Warlow & Hawton, 1989). Another reports a prevalence of 18% at six months (MacHale, O'Rourke, Wardlaw & Dennis, 1998) however, a more recent study found a prevalence of 34% (Kim & Choi-Kwon, 2000). The variations in part may be due to cultural differences or due to variations in definitions of emotional lability across studies as well as methodology employed for assessing emotional lability. Some studies use inpatients whereas others use a community based sample which may also lead to variations.

A construct that may impact on psychological distress following stroke is emotion regulation. This refers to a set of 'processes by which individuals influence which emotions they have, when they have them and how they experience and express these emotions' (Gross, 1998; p275) in relation to their goals. In order to regulate emotions, the ability to access, evaluate and understand them is important (Gratz & Tull, 2010). This definition of emotion regulation relates to the processes of recognising, monitoring, evaluating and modifying emotions (Phillips & Power, 2007), however, it is also thought that emotion

regulation can occur on a continuum from effortful and conscious to effortless and unconscious (Gross, 2002).

The process model of emotion regulation (Gross & Thomson, 2007) describes regulation as occurring at multiple points across the continuum of emotion generation. This focuses on strategies described as antecedent or response focused dependant upon the point in the emotion generative process in which they occur. The processes in the model are ongoing and each step influences subsequent steps with feedback from the emotional response impacting upon ongoing regulation (Richards & Gross, 2000).

Studies have found that emotion regulation has an impact on quality of life and is important in adapting to chronic illnesses such as rheumatoid arthritis (Van Middendorp, Geenen, Sorbi, Hox, Vingerhoets et al. 2005) multiple sclerosis (Phillips, Saldias, McCarrey, Henry, Scott et al. 2009) and kidney disease (Gillanders, Wild, Deighan & Gillanders, 2008). This is relevant to individuals with stroke given that there are known emotional and cognitive consequences following stroke which impact quality of life. With regard to rehabilitation, adjustment can be difficult and impact on quality of life, emotions and cognition (Lincoln *et al.* 2012). Damage to certain areas of the brain may result in suboptimal selection of emotion regulatory strategies resulting in negative social and psychological consequences, further impacting quality of life.

Emotions are thought to be involved in decision making, enhancing memory and facilitating interpersonal interactions (Gross & Thomson, 2007) and successful emotion regulation is important with regards to these goals. Reappraisal and suppression are two commonly studied emotion regulatory strategies and have been found to have different cognitive consequences. Suppression has been found to negatively impact upon decision making (Heilman, Crisan, Houser, Miclea & Miu, 2010) and memory in both laboratory and real life settings (Richards and Gross, 2000). Working memory capacity may facilitate emotion regulation and those with better working memory capacity may experience less negative affect in response to negative feedback (Schmeichel & Demaree, 2010). This is important with regard to individuals with stroke as if an individual has impairments in working memory, this may then negatively impact on emotion regulation.

Carter (2008) found that using strategies to regulate emotions led to increased activity in the left dorsolateral prefrontal cortex. Beer, Knight and D'Esposito (2006) found that the lateral

orbitofrontal cortex is important for assessing the contextual relevance of emotion for decision making, however, this study was not carried out for real life emotions and participants were primed with neutral and negative pictures which may not have been relevant to them. These studies may support evidence that the dorsolateral and orbitofrontal cortex are involved in emotion, however, they may play different roles. It is thought that limbic subcortical areas are involved in emotion generation and cortical areas are involved in regulation (Le Douarin, 1995) and the strategy of reappraisal is thought to occur through interactions between the prefrontal cortex and subcortical networks (Wagner, Davidson, Hughes, Lindquist & Ochsner, 2008).

With regards to cognitive functioning, the brain areas involved in emotion generation and regulation outlined above are also known to be involved in executive functions (Lincoln *et al.* 2012) including set shifting, inhibition, initiation, selective attention and mental flexibility. A review of imaging studies by Ochsner and Gross (2008) found areas of the brain implicated in selective attention, response inhibition and monitoring processes were activated during the emotion regulatory strategy of reappraisal. This has implications for the role of executive functions as reappraisal involves generating, implementing and tracking a strategy (Ochsner & Gross, 2008). This is likely to involve processes described in the Supervisory Attentional System (Shallice & Burgess, 1998). Gyurak, Goodkind, Madan, Kramer, Miller & Levenson (2009) found that high verbal fluency scores were related to greater emotion regulation, indicated by more successful expressive suppression. This highlights a link between executive functioning and emotion regulation.

The current research aims to explore the differences in emotion regulation and quality of life between a group of individuals with stroke and a control group. It also aims to explore associations between emotion regulation, quality of life, executive functioning and anxiety and depression following stroke. It was hypothesised that the individuals with stroke would have significantly more difficulties in emotion regulation and have significantly lower self-reported quality of life than the control group. With regard to correlations, it was hypothesised that greater difficulties in emotion regulation would be associated with lower self-reported quality of life and higher anxiety and depression in the stroke group. It was also hypothesised that executive dysfunction would be associated with greater difficulties in emotion regulation and lower self-reported quality of life in the stroke group.

Method

Participants

Participants were identified via the NHS Grampian stroke register with 180 participants who had a stroke between November 2010 and February 2012 identified as eligible to participate and contacted by letter. Fifty (28%) gave informed consent and participated in the study. Ethical approval was granted by the North East of Scotland Research Ethics Committee. The inclusion criteria were: diagnosis of stroke (haemorrhagic and/or ischaemic); medically stable, able to give informed consent and aged over 18. Participants were not included in the study if they had a current or previous history of any other condition known to affect cognitive functioning. Those with aphasia or any visual or hearing impairment that would affect their ability to complete questionnaires or neuropsychological assessment were not included in the study.

The control group participants were recruited from a local community group and were given the option of attending an appointment in a church hall or a home visit. A total of 45 control participants gave informed consent and participated in the study. The inclusion criteria were the same as for the stroke group other than diagnosis of stroke.

Measures

For all participants, demographic data was collected on age, gender, and years of formal education. For the individuals with stroke, information was also collected on type of stroke, location of lesion and time since stroke. Measures were administered according to manual instructions.

Three validated self report questionnaires were completed by all participants in the current study and are detailed below. The National Adult Reading Test (Nelson & Willison, 1991) was administered to all participants to control for premorbid IQ.

Difficulties in Emotion Regulation Scale (Gratz & Roemer, 2004)

This is a 36 item self report measure used to explore differences in emotion regulation difficulties between the individuals in the stroke group and the control group. It assesses six domains of emotion regulation (non acceptance of emotions, awareness of emotions, difficulties engaging in goal directed behaviour, impulse control difficulties, limited access to emotion regulation strategies and lack of emotional clarity). There is good evidence for

the validity and reliability of these subscales and these will be used to compare the participants with stroke to the control group. There is also a total score with high internal consistency (Cronbach's alpha = 0.93) and test retest reliability of 0.88 (Gratz & Roemer, 2004). This total score will also be used to compare participants in each group and will be used in the correlational analyses.

Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983)

This is a 14 item self report measure with seven items relating to anxiety and seven relating to depression. Although there are cut off scores available for a stroke population (Johnson et al, 1995; Aben et al 2002), the original cut off scores (Snaith & Zigmond, 1994) were used to compare participants as there is no consensus with regard to the optimal cut off scores for this population. This measure has been recommended for use in a stroke population without communication problems (Bennett & Lincoln, 2004) as it does not rely on physical or somatic items and so is less likely to be affected by misleadingly high scores in a population with physical illness. It is also brief and easier than other measures for stroke participants to complete (O'Rourke et al. 1998) and places low demands on memory. Cronbach's alpha for the anxiety subscale is 0.82 and 0.77 for the depression subscale (Crawford, Henry, Crombie & Taylor, 2001). The subscales will be used to compare participants in the control group to individuals with stroke and in the correlational analyses.

World Health Organization quality of life questionnaire – brief version

This is a 26 item self report measure assessing four domains of quality of life (physical health, psychological health, social relationships and environment). It was developed as a shorter version of the WHOQOL-100. Correlations between the four domain scores on the WHOQOL-100 and WHOQOL-BREF ranged from 0.89 to 0.95 and the internal consistency of the four domains on the WHOQOL-BREF ranged from 0.66 to 0.84 (Harper & Power, 1998) indicating it is an acceptable substitute. Raw scores on the WHOQOL-BREF are summed for each domain and converted to scores between 0-100 using tables provided in the manual (Harper, 1997) to allow comparison between the domains. These converted scores will be used in the analyses. For the physical health, psychological health and environment domains, Cronbach's alpha for internal consistency were 0.82, 0.81 and 0.80 respectively, however, for social relationships was 0.62 (Skevington, Lotfy & O'Connell, 2004).

National Adult Reading test (Nelson & Willison, 1991)

This was used as an indicator of premorbid IQ and the number of errors made was used to calculate the predicted full scale IQ (Nelson & Willison, 1991). This was used to compare the group of participants with stroke to the participants in the control group and to control for differences in premorbid IQ therefore all participants completed this measure. The NART allows interpretation of premorbid functioning in the context of current functioning. Crawford, Parker and Besson (1988) found a split half reliability of 0.9, inter-rater reliability between 0.96 and 0.98 and test retest reliability to be 0.98 and O'Carroll (1987) found high inter-rater reliability. Crawford, Stewart, Cochrane, Parker and Besson (1989) also found a correlation of 0.73 between IQ measured at age 11 and NART performance aged 77. The NART was therefore used in the current study to match the stroke group with the control group, however, given evidence that NART performance may be affected by neurological damage, scores were interpreted with caution.

Executive functioning measures

Four measures which seek to assess different aspects of executive functions were chosen to assess set shifting, inhibition, mental flexibility and divided attention. These were administered to participants in the stroke group only.

Brixton Spatial Anticipation Test

This was used to measure rule detection and following and set shifting. This requires the participant to detect and follow rules and then shift set and detect the new rule. The raw score is the number of errors made and this is normally converted to a Sten score, however, Crawford and Henry (2005) report that Sten scores are coarsely grained and may obscure differences in raw scores. For this reason, the current research used the number of errors made on the Brixton in the analyses and years of education was controlled for as it is known to affect scores on this measure. The split half reliability for this measure is 0.62 and test retest reliability was 0.71 (Burgess & Shallice, 1997). Reverberi, Lavaroni, Gigli, Skrap and Shallice (2005) administered this test to 40 individuals with focal frontal lobe lesions (vascular, tumour and traumatic brain injury) and 43 control participants and found that all individuals with lesions were impaired as compared to control participants.

Hayling Sentence Completion Test

This was used to measure inhibition and is associated with dysfunction in everyday life (Burgess & Shallice 1997). There are raw scores for time taken and for number of errors for

parts one and two. These are then converted to scaled scores which are summed and converted to a total scaled score. For the purposes of the current research, the total scaled score was used as a measure of inhibition. As Hayling scaled scores do not take years of education into account but are known to be affected by this, years of education was controlled for in the analysis. The test retest reliability is 0.76 for the total scaled score (Burgess & Shallice, 1997).

Verbal Fluency

Verbal fluency is a commonly used measure of mental flexibility. It consists of letter fluency and category fluency. In the letter fluency trials, participants were asked to name as many words as possible in one minute beginning with a particular letter. There were three trials of this consisting of the letters F, A and S. The total number of correct words generated over these three trials was converted to a t score using norms based on years of education (Gladsjo, Schuman, Evans, Peavy, Miller & Heaton, 1999). Category fluency involved participants generating as many animal names as possible in one minute and the total number of animals is then converted to a t score using norms based on years of education (Gladsjo et al. 1999). Henry and Crawford (2004) report that this measure has good norms and reliability as well as moderate ecological validity and moderate sensitivity.

Color Trails Test

This was developed as a more culturally fair version of the Trail Making Test thought to be less affected by cultural and linguistic bias (D'Elia, Satz, Uchiyama & White, 1996). Part one measures sustained attention and processing speed and part two measures set shifting and divided attention. The interference index was calculated by subtracting the time taken for part one from the time taken for part two and divided by the time taken for part one. This is hypothesised to give a purer measure of divided attention, by controlling for the effects of processing speed (D'Elia et al. 1996). The manual classifies scores on an ordinal scale according to the percentile range they fall into for age and years of education (<1; 2-5; 6-10; 11-16; >16). As other scores in the current study are in the form of continuous variables, raw scores were used for the interference index in the analyses. This provides a greater range of scores for analysis. As this score does not take years of education into account, this was controlled for in the analysis. The test retest reliability of part one is 0.64 and for part two it is 0.79 (D'Elia et al. 1996). The Color Trails test was found to be significantly correlated with the Trail Making Test in the normative sample and the color trails time

variables were significantly correlated with each other in normative and clinical samples (D'Elia et al. 1996).

Statistical Analysis

Hypotheses one and two involved independent samples t tests to compare the stroke group with the control group. A power calculation using GPower (Faul Erdfelder, Lang & Buchner, 2007) and assuming a medium-large effect size indicated a total sample of 90 was needed. Hypotheses three to six, involved a within subjects design to explore correlations between measures in the stroke group. A power calculation assuming a medium-large effect size indicated a sample size of 37 was needed.

Data was analysed for normality, homogeneity of variance, skew and kurtosis. The kolmogorov-smirnov test of normality was used to assess normality of the variables alongside skew, kurtosis, QQ plots and histograms. Skew and kurtosis were transformed to z scores by dividing the score by the standard error for skew and by square rooting this value for kurtosis (Field, 2005) and were assessed as significant if the score was greater than 2.58 (Clark-Carter, 1997).

Transformation of data was carried out where assumptions for parametric statistics were not met which occurred in four subscales of the DERS (goals, impulsivity, strategies, clarity). Logarithm transformation were carried out based on the distributions of the histograms (Clark-Carter, 1997) but these were unsuccessful and did not improve skew or kurtosis in either group. Non parametric statistics were consequently planned for those variables.

The physical domain of the WHOQOL-BREF did not meet the assumptions for parametric statistics therefore a successful square root transformation was carried out based on the distributions of the histograms (Clark-Carter, 1997). All domains of the WHOQOL-BREF involved in subsequent analysis with the physical domain were consequently transformed in the same way as advised by Field (2005) for comparison of the group means. This enabled parametric statistics to be used.

Independent samples t tests were used to explore the demographic differences between the stroke and control groups and the Welch statistic was reported when data did not meet the requirements for homogeneity of variance (Pallant, 2011). Independent samples t tests for

parametric data and Mann-Whitney U tests for non parametric data were planned, however, as estimated premorbid IQ (as measured by the NART) and years of education were significantly different between the control and stroke groups, they were explored as covariates. Preliminary checks were conducted to ensure no violation of the assumptions of normality, linearity, homogeneity of variances, and homogeneity of regression slopes (Pallant, 2011). Years of education was chosen as a covariate rather than NART IQ as these variables were strongly correlated with one another (Clark-Carter, 1997) and the NART IQ regression slopes did not meet the assumption of homogeneity of regression slopes. As depression and emotion regulation are theoretically linked, they will share a lot of variance, therefore the HADS depression subscale was not used as a covariate in the emotion regulation analysis.

Although there is no non parametric equivalent of ANCOVA, parametric tests have been found to be robust when some assumptions are violated (Clark-Carter, 1997) and if there is a large sample, particularly over 30 in each group, parametric statistics are more robust to violations of normality (Motulsky, 2010). The non normal data were inspected and it was found that skew was significant, however, kurtosis was not significant for these variables and the histograms did not show bimodal samples. Vickers (2005) present evidence that ANCOVA is favourable to Mann-Whitney in such cases. This approach is similar to Harrison et al. (2010) when dealing with DERS variables.

Correlational analyses

Preliminary analyses were carried out to ensure no violation of the assumptions of normality, linearity and homoscedasticity (Pallant, 2011). As multiple comparisons increase the likelihood of a type one error, a conservative p value of .01 was chosen to minimise the likelihood of significant results due to chance. This was chosen over the use of a Bonferroni correction which involves dividing the alpha level by the number of comparisons (Motulsky, 2010) and may increase the likelihood of a type two error and mask significant results. The executive functioning measure, DERS total score and three domains of the WHOQOL-Bref were normally distributed enabling parametric Pearson product moment correlations to be carried out. These were bivariate one tailed correlations. Partial correlations were carried out using years of education as a covariate for the Hayling, Brixton and Color Trails interference index correlations. The physical domain of the WHOQOL-BREF was not normally distributed therefore this domain used the non parametric Spearman's rank order correlations in the analysis.

Results

Exploratory data analysis

Independent samples t tests explored differences in key demographic variables between the two groups. The control group had significantly higher estimated premorbid IQ (NART) ($m=112.8$, $sd=8.9$) than the stroke group ($m=108.2$, $sd=10.2$; $t(93) = -2.305$, $p = .026$). The control group also had significantly more years of education ($m=14.62$, $sd=3.9$) than the stroke group ($m=12.8$, $sd=3.6$; $t(93) = 2.319$, $p=.023$). The stroke group had significantly higher levels of depression ($m=4.4$, $sd=3.5$) than the control group ($m=2.2$, $sd=1.7$; $t(72) = 4.045$, $p < .001$). There were no significant differences in age or anxiety. There were 28 (56%) males and 22 (44%) females in the stroke group ($n=50$) and 18 (40%) males and 27 (60%) females in the control group ($n=45$) though this difference was not statistically significant ($\chi^2 = 1.83$, $p=.18$).

Descriptive statistics

Using the Oxfordshire Community Stroke Project classification (Mead, Lewis, Wardlaw, Dennis & Warlow, 2000), the most common type of stroke in the current study was partial anterior circulation stroke (38%) followed by lacunar stroke (26%), posterior circulation stroke (20%), total anterior circulation stroke (10%) and haemorrhagic stroke (3%). Nineteen participants (38%) had a stroke in the right hemisphere and 30 participants (60%) in the left hemisphere. There was no information regarding hemisphere for one participant. Time since stroke ranged from one to seventeen months with a mean of 8.5 ($sd\ 4.2$) months since stroke.

HADS scores were classified as normal, mild, moderate and severe according to criteria set by Snaith and Zigmond (1994). As can be seen from Table 4.1, whilst levels of above-normal anxiety amongst stroke survivors (36%) were similar to those of age-matched controls (31%), levels of above-normal depression were higher amongst stroke survivors (22%) than controls (0%).

Table 4.1: HADS categories across groups

	HADS anxiety		HADS depression	
	Stroke	Control	Stroke	Control
Normal	32 (64%)	31 (69%)	39 (78%)	45 (100%)
Mild	14 (28%)	11 (24%)	8 (16%)	0
Moderate	2 (4%)	3 (7%)	3 (6%)	0
Severe	2 (4%)	0	0	0

Hypothesis driven results

Between subjects analyses

- 1. Participants with stroke will have significantly more difficulties in emotion regulation than control participants*

After adjusting for years of education, stroke participants had significantly higher scores for the DERS total score ($F(1,93)=4.397$, $p=.039$, partial eta squared = .046), the DERS non acceptance sub scale ($F(1,93)=7.665$, $p=.007$, partial eta squared=.077) and the DERS impulsivity subscale ($F(1,93)=4.122$, $p=.045$) indicating greater difficulties in emotion regulation. There was no significant difference for the awareness subscale ($F(1,93)=1.545$, $p=.217$) the goals subscale ($F(1,93)=2.361$, $p=.128$), the strategies subscale ($F(1,93)=2.363$, $p=.128$) or the clarity subscale ($F(1,93)=1.445$, $p=.232$). The unadjusted and adjusted means can be seen in Table 4.2.

Table 4.2 Adjusted and unadjusted means for emotion regulation subscales

	Stroke (n=50)		Control (n=45)	
	unadjusted mean (SD)	adjusted mean (SE)	unadjusted mean (SD)	adjusted mean (SE)
Awareness	15.72 (5.72)	15.49 (.76)	13.84 (4.1)	14.1 (.81)
Non acceptance	11.36 (5.38)	11.21 (.64)	8.42 (3.29)	8.59 (.68)
Impulsivity	9.6 (3.75)	8.52 (.46)	8.07 (2.44)	8.15 (.48)
Goals	10.84 (4.37)	10.84 (.57)	9.60 (3.03)	9.6 (.58)
Strategies	14.04 (6.26)	13.86 (.75)	11.96 (3.85)	12.16 (.79)
Clarity	9.34 (3.8)	9.02 (.42)	7.91 (2.61)	8.27 (.45)
Total score	69.44 (23.23)	68.7 (2.76)	59.33 (14.1)	60.16 (2.92)

2. *Participants with stroke will have significantly lower self-reported quality of life than control participants*

After adjusting for years of education, stroke participants had significantly lower scores on the physical health domain ($F(1,93)=24$, $p=.001$, partial eta squared=.207), psychological domain ($F(1,93)=3.973$, $p=.045$, partial eta squared=.041) and the environmental domain ($F(1,93)=9.079$, $p=.003$, partial eta squared=.09). There was no significant difference in the social relationships domain ($F(1,93)=2.752$, $p=.101$, partial eta squared=.029).

Correlational Analyses

3. *There will be a correlation between executive dysfunction and greater difficulties in emotion regulation amongst stroke participants.*

As can be seen in Table 4.3, although correlation coefficients were in the expected direction indicating that higher DERS (greater emotion dysregulation) scores were associated with poorer executive functioning, none of these were significant.

Table 4.3: Correlation matrix of main variables

Variable	Executive functioning measures					Emotion Regulation
	Letter	Category	Color Trails Index	Brixton	Hayling	DERS
<u>Emotion Regulation</u> DERS total score	-.20	-.25	.03	-.1	-.17	
Physical Quality of life #	.21	-.02	.37*	-.2	.29	-.58*
Psychological Quality of Life	.16	.11	.25	.06	.27	-.71*
Social Relationships Quality of life	.05	.07	.31	-.18	.26	-.62*
Environment Quality of life	.03	.2	.30	-.04	.19	-.45*

* $p<.01$; #spearman's rho, $N=50$ for all correlations;

DERS = difficulties in emotion regulation scale

4. *There will be a correlation between greater difficulties in emotion regulation and lower self-reported quality of life amongst stroke participants.*

As can be seen in Table 4.3, there were significant negative correlations between the difficulties in emotion regulation scale and all domains of quality of life, indicating that

greater difficulties in emotion regulation were significantly associated with lower self-reported quality of life. All of these correlations had a large effect size (Cohen, 1988).

5. *There will be a correlation between difficulties with executive function and lower self reported quality of life in the stroke group*

Table 4.3 shows that the physical domain of quality of life was significantly positively correlated with the color trails interference index with a medium effect size. Although not significant, all correlation coefficients between the Hayling and all domains of quality of life were in the expected direction with small effect sizes and approaching significance. There were no associations between quality of life and letter fluency, category fluency or the Brixton.

6. *There will be a correlation between emotion regulation difficulties and higher self reported anxiety and depression in the stroke group*

The relationship between anxiety and depression as measured by the HADS and emotion regulation as measured by the DERS was investigated using Pearson Product Moment Correlations. There was a positive correlation between anxiety and emotion regulation ($r=.48$, $n=50$, $p<.001$) with a medium effect size and a positive correlation between depression and emotion regulation ($r=.67$, $n=50$, $p<.001$) with a large effect size.

Discussion

Main Findings

The principal aim of the study was to determine if there were significantly greater difficulties in emotion regulation and lower self reported quality of life in individuals with stroke compared to a control group. Further aims were to determine if there were associations between executive functioning, emotion regulation and quality of life. It was predicted that stroke participants would have more difficulties with emotion regulation and lower quality of life than the control group. The results were consistent with this in that participants in the stroke group had significantly higher total scores on the DERS emotion regulation measure and on the subscales of non acceptance of emotion and impulsivity. Those in the stroke group had significantly lower self reported quality of life in the physical, psychological and environmental domains of quality of life. The stroke and control groups were well matched on age, sex and the anxiety scale of the HADS, however, they were significantly different on the depression scale of the HADS, years of education and NART IQ. There were significant correlations between greater difficulties in emotion regulation and lower self-reported quality of life and between greater difficulties in emotion regulation and higher levels of anxiety and depression. Quality of life and aspects of executive function showed significant correlations. There were no significant correlations between emotion regulation and executive functions.

Previous research has found that difficulties in emotion regulation exist in other physical health conditions including multiple sclerosis (Phillips et al. 2009) and renal disease (Gillanders et al. 2008) and the current research provides support for the hypothesis that survivors of stroke also have some difficulties in emotion regulation. The stroke survivors had particular difficulties with non acceptance of emotions and impulsivity. Non acceptance of emotions as measured by the DERS has previously been found to be a core deficit in alexithymia (Pandey et al. 2008) which may be problematic as Gratz and Tull (2010) argue that acceptance of emotions is important for successful regulation of emotions. Non acceptance of emotion may be related to poor insight or anosognosia which is a commonly reported difficulty following stroke. (Lincoln et al. 2012). The use of suppression as an emotion regulation strategy has previously been found to have negative implications for memory (Richards & Gross, 2000) and may lead to impulsive decision making (Heilman et al. 2010). Non acceptance of emotions may lead to the use of suppression as a regulatory

strategy which may be detrimental in a population that may already experience cognitive impairments.

Emotions are thought to be involved in decision making, enhancing memory and facilitating interpersonal interactions (Gross & Thomson, 2007) which has implications for both executive functioning and quality of life. In the current study, despite the lack of significance, associations between emotion regulation and executive functions were in the expected direction. The correlations between the Hayling, a measure of inhibition and the DERS total score were not significant; however, they were in the expected direction with a medium effect size. Given those in the stroke group had significantly greater difficulties in the impulsivity subscale of the DERS it may be that impulsivity is a behavioural expression of executive dysfunction. The lack of significant associations was surprising given the evidence from neuroimaging that emotion generation and regulation involves both cortical and subcortical brain areas that are also thought to be involved in executive functioning (LeDoux, 1995). It may be that the aspects of executive functioning assessed in the current study are not associated with emotion regulation but that other aspects of executive functioning are. The participants in the stroke group were not selected based on difficulties in executive functioning and many performed within expected limits. It may have been beneficial to select participants based on having executive impairments in order to find out if impairments were associated with emotion regulation difficulties. In clinical practice, it is not uncommon for individuals to perform well on measures of executive functioning yet still have real life difficulties such as multitasking or performing routines. This may account for lack of association between executive dysfunction and emotion regulation difficulties, given that some participants may have performed within expected limits on these assessments despite impairments.

Difficulties in emotion regulation were significantly negatively associated with all domains of quality of life indicating that greater difficulties in emotion regulation were associated with lower self reported quality of life. Phillips and Power (2007) found that functional emotion regulation strategies as measured by the Regulation of Emotion Questionnaire were associated with greater quality of life which is consistent with the current research. Emotions are important for facilitating decision making, preparing the motor response and assisting social interactions (Keltner & Gross, 1999) therefore, any difficulties with emotions or regulating emotions is likely to impact on quality of life. It could be expected that stroke participants would have a lower self-reported quality of life in the physical domain due to

having a physical health condition; however, quality of life was also significantly poorer for stroke survivors in the psychological and environmental domains. Individuals who habitually use suppression have been found to have more negative emotions, more rumination and lower self esteem whereas those using reappraisal have less depression, higher life satisfaction and closer relationships (Gross & John, 2003). This has implications for quality of life as the strategies used for regulating emotions have psychological and social implications. The psychological domain includes questions related to enjoyment and meaningfulness of life as well as concentration, satisfaction and negative feelings. Strategies of emotion regulation such as reappraisal may be implicated in affecting psychological quality of life. For example, reappraising the thoughts regarding having had a stroke and the associated difficulties may lead to improved quality of life than suppressing negative thoughts. The environmental domain comprises questions regarding the safety and healthiness of the physical environment, availability of money, information, leisure activities, health care and transport which all may be negatively affected following stroke due to difficulties returning to work (Intercollegiate Stroke Working Party, 2008) and driving (Lincoln et al. 20012).

Associations between the Hayling and the domains of quality of life had small effect sizes and were approaching significance. There was a significant association between the color trails interference index and the physical domain of quality of life, however, there were no other significant correlations between any quality of life domains and the other executive functioning measures. This was surprising as previous research has found associations between poorer executive function and lower quality of life (Crawford & Henry, 2004) and executive dysfunction has been found to be a significant predictor of poor functional outcome one year post stroke (Lesniack et al. 2008). This may be because better ability to engage in higher level cognitive functions may lead to higher self efficacy and therefore improvements in self reported quality of life. Although the current study did not find consistent evidence for associations between poorer executive functions and lower self-reported quality of life, early detection of executive dysfunction is important for planning appropriate cognitive rehabilitation strategies (Lesniack, Czepiel, Seniow & Czlonkowska, 2008) given that executive dysfunction can impact on other cognitive functions such as memory which may negatively impact quality of life. It may be that different aspects of executive functions differentially impact on quality of life or that the measures used were not optimal for detecting any associations.

Clinical implications

The current study shows that there are difficulties in emotion regulation in survivors of stroke and these difficulties are associated with lower self-reported quality of life and higher levels of self-reported anxiety and depression. This is important as it is already known that approx 35% of stroke survivors have depression (Hackett et al. 2005) and 22-25% have anxiety (De wit et al. 2008). Emotional lability has also been found post stroke ranging from 15% (House, Dennis, Molyneux, Warlow & Hawton, 1989) to 34% (Kim & Choi-Kwan, 2000). The current study suggests emotion regulation difficulties were strongly associated with poorer quality of life and, as such, it is important that such difficulties are addressed to prevent poor quality of life amongst stroke survivors and associated risk factors for further illness. The stroke working party advises that stroke rehabilitation services should train staff in recognising and managing emotional, communicative and cognitive problems following stroke including screening every individual for depression, anxiety and emotionalism (Intercollegiate Stroke Working Party, 2008). The current research provides evidence that it may be beneficial to also screen for difficulties with emotion regulation; however, further research is warranted in order to establish the prevalence of such difficulties within the stroke population.

Greater difficulties in emotion regulation were also significantly associated with increased anxiety and depression in the current study. It therefore may be beneficial to screen for difficulties in emotion regulation in stroke survivors in order to address such difficulties. Given a number of therapeutic approaches address emotion regulation difficulties including Acceptance and Commitment Therapy (ACT), Dialectical Behaviour Therapy (DBT) and Cognitive Behavioural Therapy (CBT) (Aldao, Nolen-Hoeksema & Schweizer, 2010) it may be that these therapies would be beneficial approaches in addressing emotion regulation difficulties post stroke. The Scottish Intercollegiate Guidelines Network recommend that principles from motivational interviewing and problem solving therapy should be incorporated into education programmes post stroke and beliefs and attitudes regarding recovery should be addressed to facilitate emotional adjustment (SIGN, 2010).

Currently, the psychological therapies matrix (Scottish Government, 2011) recommends CBT for post stroke depression; however, these guidelines do not have recommendations for anxiety or emotionalism post stroke. As non acceptance of emotions was significantly poorer in the stroke group in the current study, it may be that aspects of ACT may be appropriate for addressing such difficulties. As depression following stroke is known to

impede progress in rehabilitation (Intercollegiate Stroke Working Party, 2008) it may be that there is a similar picture for emotion regulation difficulties however, this requires further exploration. The DERS has been found to be sensitive to change over time (Fox, Axelrod, Paliwal, Sleeppe & Sinha, 2007) so it could be used to assess emotion regulation difficulties following stroke and to track the success of interventions. Broomfield et al. (2010) argue that an approach using elements of motivational interviewing, CBT, grief resolution and executive skills training is the most beneficial approach to post stroke depression and aspects of this may also be important in addressing difficulties with emotion regulation. The current study did not find evidence of associations between executive function and emotion regulation; however, Broomfield et al. (2010) suggest that difficulties in executive functioning may impede ability to engage in therapeutic approaches to address difficulties. This suggests a full assessment of cognitive and emotional difficulties is warranted in individuals following stroke.

Strengths and limitations of current study

It is a strength of the current study that a control group was used that enabled comparisons to be made in a similar age range for anxiety, depression, emotion regulation and quality of life. Much of the prior research using the DERS has used younger adults and undergraduate students as control groups and there is little research into the use of the DERS in older adult populations. As stroke occurs predominantly in those over 65 (Stroke Association, 2012), comparing the participants in the current study to previous studies with younger populations may give an inaccurate finding of the difficulties in the stroke sample, therefore, it is strength that an age matched control group was used for comparison

It is also a strength of the current study that four executive functioning measures were used, each hypothesised to measure different areas of executive functioning. Burgess (2010) argues that a minimum assessment of executive functioning should include a general measure of inhibition, executive memory abilities and multitasking ability which was addressed using the Hayling (inhibition), the Brixton and verbal fluency (executive memory) and Color Trails (multitasking); however, there are many tests of executive functioning and it may be that whilst there was no correlation between the current measures and emotion regulation, other measures may have shown different relationships. It may have been beneficial to include an assessment with greater evidence of ecological validity as previous research have found individuals with executive dysfunction may perform well on traditional neuropsychological assessments but struggle in everyday life (Evans, 1996).

It may have been beneficial to separate the stroke groups into type of stroke or hemisphere of stroke to find out if emotion regulation is more common in certain types of stroke. For example, Haring (2002) reported left hemisphere stroke is more strongly related to depression; therefore, this may also be the case for emotion regulation difficulties. In the current sample, the majority of participants had left hemisphere stroke, therefore, it may be that this accounts for the high levels of anxiety, depression and difficulties in emotion regulation. A measure to screen for cognitive impairment in other domains could have been used as this may have been a factor in difficulties with emotion regulation. The time since stroke ranged from one month to seventeen months, therefore, the results can not be generalised to a particular time period following stroke.

The control group in this sample was predominantly recruited from a church group, and individuals in this group may be more actively engaged in the community and have greater social support than individuals randomly selected. This may have impacted on their scores leading to lower self reported anxiety and depression and less difficulties with emotion regulation. There may also be a selection bias in that it may be that those with higher levels of depression were less likely to opt in. This is evident in the HADS results where there is no mild depression amongst the control group. This may have also impacted quality of life due to increased social contact through church and community group attendance; however, the effect size for the social domain was small and not significant. The largest effect size was for the physical domain which may be expected for the stroke participants due to the nature of the condition.

Some of the data did not follow a normal distribution and were non transformable, however, the NART and years of education were significantly different between groups, with the control group having slightly higher NART scores and more years of education. It was considered it would be a greater limitation to present uncontrolled analyses, therefore, in the absence of a non parametric alternative, ANCOVA was used for non normal data, controlling for years of education. Parametric tests have been found to be robust when some assumptions are violated (Clark-Carter, 1997) and if there is a large sample, particularly over 30 in each group, parametric statistics are more robust to violations of normality (Motulsky, 2010).

The NART is also thought to be sensitive to neurological dysfunction (Morris, Wilson, Dunn & Teasdale, 2005) which may go some way towards explaining why the groups were

significantly different on this measure, however, NART and years of education were found to be significantly correlated and both significantly differed between groups, therefore it may reflect a selection bias.

Future research

Given prior evidence that suppression is associated with poorer outcomes, it would be beneficial for future research to use a measure such as the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) which assesses the strategies of reappraisal and suppression. This would enable investigation into whether difficulties in emotion regulation in stroke survivors are characterised by the use of suppression which is thought to have negative consequences if used in the long term (Gross & Levenson, 1997). Strategies used to regulate emotions may be associated with aspects of executive functioning. Other cognitive difficulties following stroke could be exacerbated by emotion regulation difficulties, for example, memory has previously been found to be associated with the use of suppression (Gross & John, 2003) so future research could address the links between emotion regulation and memory following stroke. Given that 78% of stroke survivors have impairment in at least one cognitive domain (Lesniack et al. 2008), emotion regulation difficulties may exacerbate or be exacerbated by some of these difficulties which is a possible direction for future research.

The current study had a range of time following stroke from one to seventeen months and future research could address changes in the acute stage of stroke and follow this up over time as has been done with depression (Berg, Palomaki, Lehtihalmes, Lonnquist & Kaste, 2003) and emotional lability (House et al. 1989) following stroke. The DERS could be used to assess change following intervention after stroke in order to find out if interventions are successful. This may be dependant on whether emotion regulation difficulties are reactive or organic which is not addressed in the current study. Trauma is associated with emotion regulation difficulties (Amstadter & Vernon, 2008) and it could be argued that emotion regulation difficulties observed following stroke may be a consequence of having experienced a traumatic event rather than being due to organic damage. This may go some way towards accounting for why emotion regulation was not associated with executive functioning. Future research could explore the relationship between trauma and emotion regulation following stroke as well as addressing whether emotion regulation is associated with cognitive functions such as attention or memory following stroke.

4.6 Summary/conclusion

The key findings from the current study were that the stroke participants had significantly greater difficulties in emotion regulation, significantly lower self-reported quality of life and higher levels of anxiety and depression than the control group. There were significant correlations between greater difficulties in emotion regulation and lower quality of life in the stroke group with large effect sizes. There were also significant correlations between greater difficulties in emotion regulation and higher levels of anxiety and depression in the stroke group. As emotion regulation was found to impact on quality of life, it is important that such difficulties are identified and addressed following stroke. Depression is known to impede rehabilitation following stroke, and, as emotion regulation was found to be significantly associated with both anxiety and depression both in the current study and in previous research, it may be that emotion regulation difficulties exacerbate difficulties engaging in rehabilitation. Future research should explore emotion regulation over the course of recovery from stroke and its relationship with executive functions and other cognitive functions.

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Overall conclusion to thesis

There are many consequences of stroke affecting physical, cognitive and emotional domains. This thesis sought to establish by means of a systematic review and an empirical study, the extent to which issues exist within executive functioning and emotion regulation.

The systematic review in part one of the thesis indicated that executive dysfunction is common following stroke and that this is not limited to frontal lesions. This fits with current theory that multiple brain systems are involved in executive functioning. The aspects of executive functions most commonly reported as impaired were processing speed, working memory, attention and cognitive flexibility. The results of the systematic review suggest executive functions should be comprehensively assessed following stroke as traditional screening measures may not pick up these difficulties. Quantifying impairments is important for developing appropriate rehabilitation programmes for individuals and supporting professionals, families and carers.

Due to establishing that executive dysfunction occurs following different types of stroke and given the high prevalence of emotional difficulties found following stroke, it was considered appropriate to investigate whether emotion regulation, which shares overlapping neurological foundations with executive functioning and is known to underpin many emotional constructs is also impaired in stroke.

The empirical study in part two of the thesis indicated that difficulties in emotion regulation, lower self reported quality of life and higher levels of anxiety and depression characterise individuals who have had a stroke as compared to a control group. As difficulties with emotion regulation were associated with poorer quality of life and higher levels of anxiety and depression, this highlights the potential importance of screening for such difficulties following stroke. It is currently recommended that all individuals are screened for anxiety, depression and emotional lability following stroke (SIGN, 2011), however, the results of the empirical study suggest screening for emotion regulation difficulties may also be important.

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Appendices

1. 'The Clinical Neuropsychologist' journal author guidelines (systematic review)
2. SIGN 50 methodology checklist (systematic review)
3. Table of papers not included in systematic review and rationale for exclusion
4. North East of Scotland Ethics Committee Ethical Approval Letter
5. NHS Grampian Research and Development Approval Letter
6. Participant invitation letter (Stroke only)
7. Participant Information sheet (Stroke)
8. Participant Information sheet (Control)
9. Participant consent form (stroke)
10. Participant consent form (control)
11. 'Journal of Clinical and Experimental Neuropsychology' author guidelines
(empirical study journal article)

Appendix 1: The Clinical Neuropsychologist journal author guidelines

(copied and pasted)

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Reference citations within the text. Use authors' last names, with the year of publication, e.g., "(Brown, 1982; Jones & Smith, 1987; White, Johnson, & Thomas, 1990)". On first citation of references with **three to five** authors, give all names in full, thereafter use [first author] "et al.". In the references, the first **six** authors should be listed in full.

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Reference list . A full list of references quoted in the text should be given at the end of the paper in alphabetical order of authors' surnames (or chronologically for a group of references by the same authors), commencing as a new page, typed double spaced. Titles of journals and books should be given in full, e.g.:

Books: Rayner, E., Joyce, A., Rose, J., Twyman, M., & Clulow, C. (2008). *Human development: An introduction to the psychodynamics of growth, maturity and ageing* (4th ed.). Hove, UK: Routledge.

Chapter in edited book: Craik, F. I. M., Naveh-Benjamin, M., & Anderson, N. D. (1998). Encoding processes: Similarities and differences. In M. A. Conway, S. E. Gathercole, & C. Cornoldi (Eds.), *Theories of memory* (Vol. 2, pp. 61-86). Hove, UK: Psychology Press.

Journal article: Adlington, R. L., Laws, K. R., & Gale, T. M. (2009). The Hatfield Image Test (HIT): A new picture test and norms for experimental and clinical use. *Journal of*

Tables

These should be kept to the minimum. Each table should be typed double spaced on a separate page, giving the heading, e.g., "Table 2", in Arabic numerals, followed by the legend, followed by the table. Make sure that appropriate units are given. Instructions for placing the table should be given in parentheses in the text, e.g., "(Table 2 about here)".

Figures

Figures should only be used when essential and the same data should not be presented both as a figure and in a table. Where possible, related diagrams should be grouped together to form a single figure. Each figure should be on a separate page, not integrated with the text. The figure captions should be typed in a separate section, headed, e.g., "Figure 2", in Arabic numerals. Instructions for placing the figure should be given in parentheses in the text, e.g., "(Figure 2 about here)".

For more detailed guidelines see [Preparation of Figure Artwork](#) .

Statistics

Results of statistical tests should be given in the following form:

"... results showed an effect of group, $F(2, 21) = 13.74$, $MSE = 451.98$, $p < .001$, but there was no effect of repeated trials, $F(5, 105) = 1.44$, $MSE = 17.70$, and no interaction, $F(10, 105) = 1.34$, $MSE = 17.70$."

Other tests should be reported in a similar manner to the above example of an F -ratio. For a fuller explanation of statistical presentation, see the *APA Publication Manual* (6th ed.).

Abbreviations

Abbreviations that are specific to a particular manuscript or to a very specific area of research should be avoided, and authors will be asked to spell out in full any such abbreviations throughout the text. Standard abbreviations such as RT for reaction time, SOA for stimulus onset asynchrony or other standard abbreviations that will be readily understood by readers of the journal are acceptable. Experimental conditions should be named in full, except in tables and figures.

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Appendix 2: SIGN methodology checklist 3 Cohort studies

(copied and pasted from SIGN 50: A guideline developers handbook)

 SIGN	Methodology Checklist 3: Cohort studies	
Study identification (<i>Include author, title, year of publication, journal title, pages</i>)		
Guideline topic:		Key Question No:
<p>Before completing this checklist, consider:</p> <ol style="list-style-type: none"> 1. Is the paper really a cohort study? If in doubt, check the study design algorithm available from SIGN and make sure you have the correct checklist. 2. Is the paper relevant to key question? Analyse using PICO (Patient or Population Intervention Comparison Outcome). IF NO REJECT (give reason below). IF YES complete the checklist.. 		
Reason for rejection: 1. Paper not relevant to key question <input type="checkbox"/> 2. Other reason <input type="checkbox"/> (please specify):		
Checklist completed by:		
Section 1: Internal validity		
<i>In a well conducted cohort study:</i>		In this study the criterion is:
The study addresses an appropriate and clearly focused question.	Well covered Adequately addressed Poorly addressed	Not addressed Not reported Not applicable
SELECTION OF SUBJECTS		
The two groups being studied are selected from source populations that are comparable in all respects other than the factor under investigation.	Well covered Adequately addressed Poorly addressed	Not addressed Not reported Not applicable
The study indicates how many of the people asked to take part did so, in each of the groups being studied.	Well covered Adequately addressed Poorly addressed	Not addressed Not reported Not applicable
The likelihood that some eligible subjects might have the outcome at the time of enrolment is assessed and taken into account in the analysis.	Well covered Adequately addressed Poorly addressed	Not addressed Not reported Not applicable

What percentage of individuals or clusters recruited into each arm of the study dropped out before the study was completed.		
Comparison is made between full participants and those lost to follow up, by exposure status.	Well covered Adequately addressed Poorly addressed	Not addressed Not reported Not applicable
ASSESSMENT		
The outcomes are clearly defined.	Well covered Adequately addressed Poorly addressed	Not addressed Not reported Not applicable
The assessment of outcome is made blind to exposure status.	Well covered Adequately addressed Poorly addressed	Not addressed Not reported Not applicable
Where blinding was not possible, there is some recognition that knowledge of exposure status could have influenced the assessment of outcome.	Well covered Adequately addressed Poorly addressed	Not addressed Not reported Not applicable
The measure of assessment of exposure is reliable.	Well covered Adequately addressed Poorly addressed	Not addressed Not reported Not applicable
Evidence from other sources is used to demonstrate that the method of outcome assessment is valid and reliable.	Well covered Adequately addressed Poorly addressed	Not addressed Not reported Not applicable
Exposure level or prognostic factor is assessed more than once.	Well covered Adequately addressed Poorly addressed	Not addressed Not reported Not applicable
CONFOUNDING		
The main potential confounders are identified and taken into account in the design and analysis.	Well covered Adequately addressed Poorly addressed	Not addressed Not reported Not applicable
STATISTICAL ANALYSIS		
Have confidence intervals been provided?		

SECTION 2: OVERALL ASSESSMENT OF THE STUDY

How well was the study done to minimise the risk of bias or confounding, and to establish a causal relationship between exposure and effect?

Code ++, +, or –

Taking into account clinical considerations, your evaluation of the methodology used, and the statistical power of the study, are you certain that the overall effect is due to the study intervention?

Are the results of this study directly applicable to the patient group targeted in this guideline?

Notes. Summarise the authors conclusions. Add any comments on your own assessment of the study, and the extent to which it answers your question.

Appendix 3: Table of papers not included in systematic review and rationale for exclusion

Paper	Primary reason for exclusion
Fellows (2006)	Stroke data not presented separately, lesions other than stroke (e.g. glioma) included.
Fellows and Farrah (2006)	Stroke data not presented separately – glioma included.
Yochim, Baldo, Nelson & Delis (2007)	Stroke data not presented separately – included surgery, cyst, meningioma and stroke.
Zanini (2008)	TBI and stroke included together – results not separated out.
Macpherson, Phillips, Della Salla & Cantagallo (2009)	Only four stroke participants included in the sample and not included separately.
Baldo & Shimamura (1998)	Mixed aetiologies – stroke not presented separately.
Sachdev, Brodaty, Valenzuela, Lorentz, Looi <i>et al.</i> (2004)	Stroke and TIA presented together instead of as separate groups.
Viscogliosi, Belleville, Desrosiers, Caron & Ska (2011)	Reports correlations between executive functions and other measures rather than rates of impairment, scores are not able to be extracted.
Pahlman, Gutierrez-Perez, Savborg, Knopp & Tarkowski (2011)	Provides correlations between executive functions and other measures rather than rates of impairment in a stroke population.
Jovanovski (2010)	Developing new measure, using TBI and stroke.
Park, Lombardi, Tarita-Nistor, Roy, Gravely & Black (2012)	Provides correlations between executive functions and other measures rather than rates of impairment in a stroke population.
Grau-Olivares, Arboix, Junque, Arenaza-Urquijo, Rovira & Bartres-Faz (2010)	Studying grey matter atrophy and white matter lesions rather than stroke.
Burton, Kenny, O'Brien, Stephens, Bradbury <i>et al.</i> (2004)	White matter hyperintensities rather than stroke.
Rousseaux, Castelnat, Rigaux, Kozlowski & Danze (2009)	Study of locked in syndrome which is likely to be different from other stroke.
Piamarta, Iurlaro, Isella, Atzeni, Grimaldi <i>et al.</i> (2004)	No suitable control group.
Nguyen, Evans & Zonderman (2007)	Although different medical conditions were included, a suitable control group was not used

Appendix 4: North East of Scotland Ethics Committee Ethical Approval Letter

NRES Committees - North of Scotland

Summerfield House
2 Eday Road
Aberdeen
AB15 6RE

Telephone: 01224 558474
Facsimile: 01224 558609
Email: nosres@nhs.net



14 December 2011

Mrs Mhairi Yule
Department of Neuropsychology
Room 2.21
Ashgrove House
Foresterhill
ABERDEEN
AB25 2ZN

Dear Mrs Yule

Study title:	Emotion regulation, executive function and quality of life in people who have had a stroke
REC reference:	11/NS/0041
Protocol number:	N/A

Thank you for your letter of 14 December 2011 responding to the Committee's request for further information on the above research and submitting revised documentation.

The further information was considered by the Scientific Officer.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised, subject to the conditions specified below.

Ethical review of research sites

NHS sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion" below).

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission ("R&D approval") should be sought from all NHS organisations involved in the study in accordance with NHS research governance arrangements.

Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at <http://www.rdforum.nhs.uk>.

It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document	Version	Date
Covering Letter		05 December 2011
Investigator CV		10 October 2011
Letter of invitation to participant	1	14 December 2011
Other: Paul Morris - CV		10 October 2011
Other: Reminder Letter of Invitation	1	05 December 2011
Other: Correspondence from the Caldicott Guardian		08 December 2011
Participant Consent Form: Stroke Sample	2	25 November 2011
Participant Consent Form: Control Group	1	05 December 2009
Participant Information Sheet: Stroke Sample	2	25 November 2011
Participant Information Sheet: Control Group	1	05 December 2011
Protocol	1.1	10 October 2011
Questionnaire: Hospital Anxiety and Depression Scale (HADS)		10 October 2011
Questionnaire: DERS		10 October 2011
Questionnaire: Quality of Life		10 October 2011
REC application	3.3	06 October 2011
Response to Request for Further Information		

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Reporting requirements

The attached document "*After ethical review – guidance for researchers*" gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Notification of serious breaches of the protocol
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

Feedback

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

Further information is available at National Research Ethics Service website > After Review

11/NS/0041

Please quote this number on all correspondence

With the Committee's best wishes for the success of this project

Yours sincerely

pp *RSivabanes*
Professor Siladitya Bhattacharya
Chair

Enclosures: After ethical review – guidance for researchers

Copy to: University of Edinburgh
NHS Grampian R&D Department

Appendix 5: Letter from Research & Development

Research and Development

Foresterhill House Annexe
Foresterhill
Aberdeen
AB25 2ZB



Mrs Mhairi Yule
Dept of Neuropsychology
Room 2.21
Ashgrove House
Foresterhill
Aberdeen
AB25 2ZN

Date 16/01/2012
Your Ref
Our Ref 2011PC007

Enquiries to
Extension 51121
Direct Line 01224 551121

Dear Mrs Yule

Management Permission for Non-Commercial Research

MREC Ref: N/A
NOSRES Ref: 11/NS/0041
NRS Ref: N/A
Project title: Emotion regulation, executive function and quality of life in people who have had a stroke.

Thank you very much for sending all relevant documentation. I am pleased to confirm that the project is now registered with the NHS Grampian Research & Development Office. The project now has R & D Management Permission to proceed locally. This is based on the documents received from yourself and the relevant Approvals being in place.

All research with an NHS element is subject to the Research Governance Framework for Health and Community Care (2006, 2nd edition), and as Chief or Principal Investigator you should be fully committed to your responsibilities associated with this.

It is particularly important that you inform us when the study terminates.

The R&D Office must be notified immediately and any relevant documents forwarded to us if any of the following occur:

- A change of Principal Investigator, Chief Investigator or any additional research personnel
- Premature project termination
- Any amendments – substantial or non-substantial (particularly a study extension)
- Any change to funding or any additional funding

We hope the project goes well, and if you need any help or advice relating to your R&D Management Permission, please do not hesitate to contact the office.

Yours sincerely

A handwritten signature in black ink, appearing to be 'S. Ridge', with a stylized flourish at the end.

Susan Ridge
Non-Commercial Manager

NHS Grampian

Department of Neuropsychology
Room 2.19 Ashgrove House
Aberdeen Royal Infirmary
Aberdeen AB25 2NZ AB25 2NZ
Secretary 01224-559352
Direct 01224-553399

Letter of Invitation Emotion Regulation in stroke

Dear

You are being invited to take part in a research study about the relationship between controlling emotions and recovery from stroke. . You have been contacted because your Stroke Consultant thought you would be appropriate to be contacted about the study and because your details are on the NHS Grampian Stroke Database.

Attached to this letter is an information sheet, explaining why the study is being done and what it involves. After reading the Information Sheet, if you decide you are interested in hearing more about the study and may wish to take part, please complete the enclosed Contact Sheet and return it using the stamped, addressed envelope provided.

Once the contact sheet is received you will be telephoned within one week to ask whether you are interested in taking part.

If you have any questions please do not hesitate to contact us.

Yours sincerely

Dr Mary-Joan MacLeod
Lead Stroke Consultant

Mhairi Yule
Trainee Clinical Psychologist
Room 2.19 Ashgrove House
ARI
Foresterhill
Aberdeen
Tel: 01224 553399
Mhairi.yule@nhs.net

Dr Jackie Hamilton
Clinical Neuropsychologist
Room 2.19 Ashgrove House
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Foresterhill
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Tel: 01224 553399
jackie.hamilton@nhs.net

Participant Information Sheet
(version 2; 25/11/11)
Stroke sample



1. Study title

Regulating emotions, quality of life and general thinking skills in people who have had a stroke.

2. Invitation Paragraph

You are being invited to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether you wish to take part. Thank you for reading this.

3. What is the purpose of this study?

As part of my Doctorate training in Clinical Psychology I am carrying out a study that aims to develop a greater understanding of how people who have had a stroke deal with emotions. This information is important because difficulties with regulating emotions have previously been found to be linked to depression. This could have an impact on rehabilitation goals after stroke. The information provided by participants will be used to help develop knowledge in this area and may influence rehabilitation interventions. The study plans to find out if there is a link between difficulties regulating emotions, quality of life and general thinking skills. The study will run until October 2012.

4. Why have I been chosen?

In order to investigate these issues we would like to recruit a group of people over the age of eighteen years old who have had a stroke. You have been identified through the NHS Grampian stroke database and you are being asked to take part because you meet the criteria. We plan to have approximately 45 people who have had a stroke take part in this study.

5. Do I have to take part?

No. It is up to you to decide whether to take part. If you do decide to take part, you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part, you are still free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part, will not affect the standard of care you receive.

6. What will happen to me if I take part?

You would be asked to attend one appointment which should last no longer than an hour and a half. Alternatively, you can choose to be seen over two separate appointments if you would prefer. You will be offered an appointment to come to a clinic within NHS Grampian. Alternatively you can choose for the researcher to visit you at home if you would prefer. Unfortunately it is not possible to reimburse any travelling expenses.

You will be asked to complete one questionnaire about how you control your emotions and one about your view on your quality of life. You will also be asked to complete a questionnaire looking at anxiety and depression. You will be asked to complete 4 separate tests which measure your general thinking skills. You will not be asked to complete any other tasks out with this appointment.

7. What are the possible disadvantages and risks of taking part?

There are no known risks associated with taking part in this research study. During the assessment appointment, if you become tired or feel under stress, you are free to stop at any time, without providing a reason.

If we identify high scores on measures of anxiety or depression we will encourage you to discuss this with your GP.

8. What are the possible benefits of taking part?

There are no known immediate benefits to taking part in the research study. We hope that this study will provide useful information relating to emotion regulation after stroke which may assist in developing rehabilitation interventions. This will hopefully benefit others who have had a stroke.

9. What if something goes wrong?

There are no risks associated with taking part in this research, but, if you wish to complain or have any concerns about the way the research is being conducted or any aspect of the way you have been approached or treated during the course of this study, the normal National Health Service complaints mechanisms should be available to you.

10. Will my taking part be kept confidential?

All information collected about you during the course of the research will be anonymised and kept strictly confidential. Other parties may be informed if further information emerges that raises serious concerns about your health and wellbeing, (for example, your GP) however, should this happen, the researcher will discuss this fully with you.

11. What will happen to the results of the study?

The results from this study will be written up and submitted for academic review through the University of Edinburgh Clinical Psychology Doctoral course. I will send you a summary of the main results of the study if you would like to receive this. No participants will be identified in any publication or presentation.

12. Who is organising and funding the research?

This research is jointly organised by NHS Grampian and the University of Edinburgh. The research is being carried out as part of my Doctorate in Clinical Psychology and is not being funded.

13. Who has reviewed the study?

This study has been reviewed and approved by the NRES committees - North of Scotland. The study will also be reviewed on a regular basis by supervisors within the Clinical and Health Psychology Department at the University of Edinburgh and NHS Grampian.

14. Contact for further information

Should you require any further information please do not hesitate to contact me: Mhairi Yule, Department of Neuropsychology, room 221, Ashgrove House, Foresterhill, Aberdeen, AB25 2ZH or by telephone on 01224 553399, a message can be left on this number if I am unavailable or by email on mhairi.yule@nhs.net You can also contact Dr Jackie Hamilton, Clinical Neuropsychologist, Research Supervisor, at Department of Clinical Neuropsychology, room 221, Ashgrove House, Foresterhill, Aberdeen, AB25 2ZH. Jackie.hamilton@nhs.net

Thank you for considering taking part in this study.

Participant Information Sheet – Control Group

(version 1: 5/12/11)



1. Study title

Emotion Regulation, quality of life and general thinking skills in people who have had a stroke.

2. Invitation Paragraph

You are being invited to take part in a research study. Before you decide whether or not to take part, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether you wish to take part. **You have been invited to take part because you have not had a stroke.** Learning more about how people who have not had a stroke perform these tasks is also helpful.

Thank you for reading this.

3. What is the purpose of this study?

As part of my Doctorate training in Clinical Psychology I am carrying out a study that aims to develop a greater understanding of how people who have had a stroke deal with emotions. This information is important because difficulties with regulating emotions have previously been found to be linked to depression. This could have an impact on rehabilitation goals after stroke. The information provided by participants will be used to help develop knowledge in this area and may influence rehabilitation interventions. The study plans to find out if there is a link between difficulties regulating emotions, quality of life and general thinking skills. The study will run until October 2012.

4. Why have I been chosen?

You have been invited to take part because you have not had a stroke.

Learning more about how people who have not had a stroke perform these tasks is also helpful. We will be comparing the results of people who have had a stroke to those who have not had a stroke to look at where the differences are.

5. Do I have to take part?

No. Taking part is entirely voluntary. If you decide not to take part, you do not have to give a reason. Similarly, if you agree to participate, I would also like to inform you that you are free to withdraw from this study at any time without having to give a reason. This will not affect possible future care or treatment.

6. What will happen to me if I take part?

You would be asked to attend one appointment which should last no longer than 45 minutes. You will be offered to be seen at clinic, at home or after church in the church hall. You will be asked to complete one questionnaire about how you control your emotions and one about your view on your quality of life. You will also be asked to complete a questionnaire looking at anxiety and depression. You will be asked to read out a list of words. You will not be asked to complete any other tasks out with this appointment. One of the questionnaires you will be asked to complete is designed to help identify individuals who are depressed. Scoring highly on this questionnaire does not necessarily mean that you are depressed. However, if you were found to score within the depressed range, it would be good practice for me to advise you to consult with your General Practitioner

7. Will my taking part be kept confidential?

All information, collected about you during the course of the research will be anonymised and kept strictly confidential. Other parties may be informed if further information emerges that raises serious concerns about your health and wellbeing, (for example, your GP) however, should this happen, the researcher will discuss this fully with you.

8. What will happen to the results of the study?

The results from this study will be written up and submitted for academic review through the University of Edinburgh Clinical Psychology Doctoral course. I will send you a summary of the main results of the study if you would like to receive this. No participants will be identified in any publication or presentation.

9. Who is organising and funding the research?

This research is jointly organised by NHS Grampian and the University of Edinburgh. The research is being carried out as part of my Doctorate in Clinical Psychology and is not being funded.

10. Who has reviewed the study?

This study has been reviewed and approved by the NRES committees - North of Scotland. The study will also be reviewed on a regular basis by supervisors within the Clinical and Health Psychology Department at the University of Edinburgh and NHS Grampian.

11. Contact for further information

Should you require any further information please do not hesitate to contact me: Mhairi Yule, Department of Neuropsychology, room 221, Ashgrove House, Foresterhill, Aberdeen, AB25 2ZH or by telephone on 01224 553399, a message can be left on this number if I am unavailable.
Or by email on mhairi.yule@nhs.net

You can also contact Dr Jackie Hamilton, Clinical Neuropsychologist, Research Supervisor, at Department of Clinical Neuropsychology, room 221, Ashgrove House, Foresterhill, Aberdeen, AB25 2ZH.

Appendix 9: Participant consent form (stroke)

Version 2 (25/11/11)

Stroke sample



Mhairi Yule

Trainee Clinical Psychologist
Department of Neuropsychology
Room 221 Ashgrove House
Foresterhill
Aberdeen AB25 2ZN
Telephone 01224 553399
E-mail mhairi.yule@nhs.net

CONSENT FORM

Title of Project: Emotion regulation, quality of life and executive function in stroke

Name of Researcher: Mhairi Yule

Please initial box

1. I confirm that I have read and understand the information sheet ☐
2. I have had the opportunity to consider this information, ask questions about it and had these questions answered satisfactorily ☐
3. I understand my participation is voluntary and I am free to withdraw at any time without giving any reason and without my medical care or legal rights being affected ☐
4. I understand that relevant sections of my medical notes and data collected during the study may be looked at by individuals from regulatory authorities or from the NHS where it is relevant to my taking part in the research. I give permission for these individuals to have access to my medical notes. ☐
5. I agree to take part in the above study ☐

Name of Patient

Date

Signature

Researcher

Date

Signature

Appendix 10: Participant consent form (control)

Version 1 (5/12/11)

Control group



Mhairi Yule

Trainee Clinical Psychologist
Department of Neuropsychology
Room 221 Ashgrove House
Foresterhill
Aberdeen AB25 2ZN
Telephone 01224 553399
E-mail mhairi.yule@nhs.net

CONSENT FORM

Title of Project: Emotion regulation, quality of life and executive function in stroke

Name of Researcher: Mhairi Yule

Please initial box

1. I confirm that I have read and understand the information sheet ☐
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Name of Participant

Date

Signature

Researcher

Date

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Appendix 11: Journal of Clinical and Experimental Neuropsychology author guidelines

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Instructions for authors

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References

Reference citations within the text. Use authors’ last names, with the year of publication, e.g., “(Brown, 1982; Jones & Smith, 1987; White, Johnson, & Thomas, 1990)”. On first citation of references with **three to five** authors, give all names in full, thereafter use [first author] “et al.”. In the references, the first **six** authors should be listed in full.

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Books: Rayner, E., Joyce, A., Rose, J., Twyman, M., & Clulow, C. (2008). *Human development: An introduction to the psychodynamics of growth, maturity and ageing* (4th ed.). Hove, UK: Routledge.

Chapter in edited book: Craik, F. I. M., Naveh-Benjamin, M., & Anderson, N. D. (1998). Encoding processes: Similarities and differences. In M. A. Conway, S. E. Gathercole, & C. Cornoldi (Eds.), *Theories of memory* (Vol. 2, pp. 61–86). Hove, UK: Psychology Press.

Journal article: Adlington, R. L., Laws, K. R., & Gale, T. M. (2009). The Hatfield Image Test (HIT): A new picture test and norms for experimental and clinical use. *Journal of Clinical and Experimental Neuropsychology*, 31, 731-753. doi:10.1080/13803390802488103

Tables

These should be kept to the minimum. Each table should be typed double spaced on a separate page, giving the heading, e.g., "Table 2", in Arabic numerals, followed by the legend, followed by the table. Make sure that appropriate units are given. Instructions for placing the table should be given in parentheses in the text, e.g., "(Table 2 about here)".

Figures

Figures should only be used when essential and the same data should not be presented both as a figure and in a table. Where possible, related diagrams should be grouped together to form a single figure. Each figure should be on a separate page, not integrated with the text. The figure captions should be typed in a separate section, headed, e.g., "Figure 2", in Arabic numerals. Instructions for placing the figure should be given in parentheses in the text, e.g., "(Figure 2 about here)".

For more detailed guidelines see [Preparation of Figure Artwork](#) .

Statistics

Results of statistical tests should be given in the following form:

"... results showed an effect of group, $F(2, 21) = 13.74$, $MSE = 451.98$, $p < .001$, but there was no effect of repeated trials, $F(5, 105) = 1.44$, $MSE = 17.70$, and no interaction, $F(10, 105) = 1.34$, $MSE = 17.70$."

Other tests should be reported in a similar manner to the above example of an F -ratio. For a fuller explanation of statistical presentation, see the *APA Publication Manual* (6th ed.).

Abbreviations

Abbreviations that are specific to a particular manuscript or to a very specific area of research should be avoided, and authors will be asked to spell out in full any such abbreviations throughout the text. Standard abbreviations such as RT for reaction time, SOA for stimulus onset asynchrony or other standard abbreviations that will be readily understood by readers of the journal are acceptable. Experimental conditions should be named in full, except in tables and figures.

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